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| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| viscosityFL_scan   * **Viscosity varies with samples**   + **variations in species**   + **variations in proteins and RBC** * **Temperature dependent**   + **decrease with increasing T**   + **Blood**   + **a non-Newtonian fluid at low shear rates (the agreggates of RBC)**   + **a Newtonian fluid above shear rates of 50 s-1**   + **Casson’s equation**   January 2008  **7**  Fahraeus-Lindqvist effect  In small tubes the blood viscosity has a very low value because of a cell-free zone near the wall.  non-newtonian_scan    **Blood Flow**   |  |  | | --- | --- | | **sigma effect**--> **Fahraeus-Lindqvist effect** | [medical dictionary](http://www.mondofacto.com/dictionary/medical.html) |   The [decrease](http://www.mondofacto.com/facts/dictionary?decrease) in [apparent viscosity](http://www.mondofacto.com/facts/dictionary?apparent+viscosity) that occurs when a [suspension](http://www.mondofacto.com/facts/dictionary?suspension), such as [blood](http://www.mondofacto.com/facts/dictionary?blood), is made to [flow](http://www.mondofacto.com/facts/dictionary?flow) [through](http://www.mondofacto.com/facts/dictionary?through) a [tube](http://www.mondofacto.com/facts/dictionary?tube) of smaller [diameter](http://www.mondofacto.com/facts/dictionary?diameter); observed in tubes [less](http://www.mondofacto.com/facts/dictionary?less) than about 0.3 [mm](http://www.mondofacto.com/facts/dictionary?mm) in diameter  Both POISEUILLE and CASSON laws describe the behavior of Newtonian fluid ( viscous)  According to the shear , stream organization and changes. Casson law takes in account the Sigma effect ( Fahraeus-Lindqvist effect ) defined as the [decrease](http://www.mondofacto.com/facts/dictionary?decrease) in [apparent viscosity](http://www.mondofacto.com/facts/dictionary?apparent+viscosity) that occurs when a [suspension](http://www.mondofacto.com/facts/dictionary?suspension), such as [blood](http://www.mondofacto.com/facts/dictionary?blood), is made to [flow](http://www.mondofacto.com/facts/dictionary?flow) [through](http://www.mondofacto.com/facts/dictionary?through) a [tube](http://www.mondofacto.com/facts/dictionary?tube) of smaller [diameter](http://www.mondofacto.com/facts/dictionary?diameter); observed in tubes [less](http://www.mondofacto.com/facts/dictionary?less) than about 0.3 [mm](http://www.mondofacto.com/facts/dictionary?mm) in diameter. PRACTICALLY, both laws , with different values, relate resistant effect of the viscosity to the flow. Casson law is just a Poiseuille law modification in particular conditions and its knowledge is not crucial in understanding venous hemodynamics.  We c a n d e f i n e a C a s s o n f luid as a s h e a r t h i n n i n g l iquid w h i c h h a s inf ini t e v i s c o s i t y at z e r o r a t e o f shear, a y i e l d stress b e l o w w h i c h n o f low o c c u r s a n d z e ro v i s c o s i t y at inf ini t e r a t e o f shear  Dear BBLee, let me answer all-in-one your always pertinent questions.  1- Reflux and below knee perforators. I didn’t “change “ the usual reflux definition when writing “Few below knee perforators show a systolic and/or diastolic reflux” because the usual reflux definition is a flow opposite to the physiologic, up to down in deep and superficial veins of the limbs and from deep veins to superficial in perforators, SFJ and SPJ. If not so, could you remember me the Sumner-Strandness definition and how did they prove it?  2- Dr Ermini , sometimes with me, sends videos in order to show evidences. This is a tough work…I’m sure that anybody would understand the concepts we try to explain if they would more or less the effort to understand. We have nothing to sale. No problem in Spain where these concepts are taught to the students in vascular Surgery nor to surgeons, students and angiologists who attend our classes in France and Italy. I feel responsible for what I have to communicate, and not for who doesn’t want to understand. A normal brain is sufficient...if the will helps it.  3- About deep shunts disconnection: Step 3 is easy and feasible alone, but in order to ”protect” it from a high overlying column ( recanalization, by-pass) , a first step (step 2 of the diagram) is performed if the conditions are correct. These conditions are the presence of a competent collateral vein to the refluxing ( shunting) superficial femoral vein is large enough ( 2d twin Superficial femoral vein or deep femoral vein well connected to the popliteal vein ). This step 2 is easy to perform and efficient ( functionally comparable to common femoral valve repair/prosthesis) because the competent valve is rather at the same level. Step 3 is performed alone when there is no deep femoral superficial closed shunt above(no competent collateral or poor connection deep femoral vein- popliteal vein). After the operation, patients are submitted to an efficient anticoagulation for 3 months. We don’t operate patients who show a deep hemodynamically significant obstruction ( attested by pressure measurement) .The conditions for these procedures are fulfilled in most patients affected of deep venous incompetence leading to ulcer. Particularly at the calf level. The low number of operations is due to that we operate only patients who resist to compression/dressing.  4- Closed angulation relates to Pitot’s tubes ( shown in our book “Principles of venous hemodynamics”) that show the flow/pressure distribution in tubes according to their angulation with the stream direction. In addition, an incompetent perforator doesn’t (cannot) show a diastolic reflux into the competent superficial veins. But it I can reflux during the systole and feed the superficial veins in the physiological direction, especially if its angulation with the deep veins flow direction where it connects is favorable. In that condition, the strong and repeated muscular contraction( sportsmen) will overload the superficial veins and enlarge them (varicose).  Très amicalement  **Shunt** is a fluid mechanics term that indicates a connection used as an alternative path between parts of a circuit. Its 2 ends are called “escape point” where the flow comes from and “re-entry point” where it redirects it. Spontaneous Palma is an alternate path to the blocked iliac, that flows from the common femoral vein ipsilateral to the blocked iliac vein towards the opposite common femoral vein through the SFJs. The ipsilateral SFJ is the escape point and the opposite one is the re-entry point. The alternative path is made of the ipsilateral great saphenous arch and it’s descending tributary ( usually external pudendal) that flow retrograde ( reflux) towards the opposite antegrade great saphenous arch and SFJ. This flow is continuous and increased by the muscular pump systole. This configuration fits to the pattern of Open Vicarious Shunt because in open circuit ( no closed circuit no recirculation) and vicarious because compensatory. OVS can be seen in other configurations.  Sometimes, spontaneous Palma OVS is combined with a GSV trunk a reflux ipsilateral to the blocked iliac vein. In that case, OVS is mixed with a closed shunt ( CS). In these mixed shunt configurations. It’s the case when a CS and OVS share the same outwards escape point fed by a deep vein , flow into an initial common venous portion , then divert in 2 branches , the CS one that connects back to a distal inwards re-entry, and the other one connects proximally into the deep veins through a specific inwards re-entry.Some times, a superficial venous shunt achieves 2 combined distinct functions say CS and OVS. The CS function is activated during the MP diastole and the OVS function is activated during the systole. In that case, CS and OVS share the same escape point , i.e the SFJ GSV , while CS drains beyond the arch down to a calf re-entry and OVS connects to the opposite femoral vein through the spontaneous Palma and the opposite GSV arch. These features must be well known in order to avoid to impair the OVS function when treating the CS. Particularly sparing both GSV arches in case of spontaneous Palma. - These examples show how much necessary is the analysis of each topographic and hemodynamic configuration to achieve a correct assessment.  For more informations: Principles of Venous hemodynamics. C.Franceschi, P.Zamboni Novapublishers. Com. New York.  , . It includes any deep or superficial venous alternative conduit why collaterals relate usually only to dilated small veins. So, a shunt is called “ open vicarious” OVS when by-passing an obstacle , “closed” CS when achieving a closed circuit and “open deviated” ODS when achieving a flow deviation in absence of obstacle or closed circuit. A classification has been proposed according to their hemodynamic function and topography  Spontaneous Palma can be called  Spontaneous Palma can be called  **An other example is a spontaneous Palma combined to a GSV ipsilateral to the iliac vein block. CS and OVS share the same escape point , i.e the SFJ GSV , while CS drains beyond the arch down to a calf re-entry and OVS connects to the opposite femoral vein through the spontaneous Palma and the opposite GSV arch**  **proprioceptor**       **n**   (Physiol)   any receptor (as in the gut, blood vessels, muscles, etc.) that supplies information about the state of the body neuromuscular receptors that register stimuli, such as stretch, tonicity, and movement within muscle  **Proprioceptor:**  A sensory receptor, found chiefly in muscles, tendons, joints, and the inner ear, that detects the motion or position of the body or a limb by responding to stimuli arising within the organism.  **Proprioceptive**  Pertaining to proprioception, or the awareness of posture, movement, and changes in equilibrium and the knowledge of position, weight, and resistance of objects as they relate to the body  **proprioceptive (prō´preōsep´tiv),**  *adj* describes the body's ability to sense the movement and position of muscles without visual guides. It is essential for any activity requiring hand-eye coordination.  Mosby's Dental Dictionary, 2nd edition. © 2008 Elsevier, Inc. All rights reserve  **proprioceptive deficit**  a defect of proprioception in which the animal acts as though it does not know where its feet are (in contrast to a cerebellar defect when the feet do not end up where the animal appears to intend that they should go).  **proprioceptive positioning**  positioning of the limbs or head and neck in response to proprioceptive inputs. The basis of postural reflexes.  **proprioceptive reflex**  a reflex that is initiated by stimuli arising from some function of the reflex mechanism itself  P**roprioceptive reflex**  one initiated by a stimulus to a proprioceptor.  **proprioceptive reflexes**  **Type:** Term  **Definitions:** 1. any reflex brought about by stimulation of proprioceptor   |  | | --- | |  | |  | "claude.franceschi@gmail.com" <claude.franceschi@gmail.com> | |  | Erika Mendoza <Erika.Mendoza@t-online.de>,  "Lattimer, Christopher" <c.lattimer09@imperial.ac.uk> |  7F766D9DE3225112FFF77A1C4Efti/kyve4rv73ry6akDear ChristopherDo you refer to the primary Chap 5 b draft or to the corrected one after the Marianne’s, Marza and Oscar comments? You suggest me to transfer some of figures and text into the other 2,6 chapters. How? So far I didn’t receive any news from the Chap 2 and 6 final writers and contributors., What a challenge. As usual in Books, Articles and Congresses, we are submitted to the the Procrustes’ rule ( In Greek mythology Procrustes made his victims fit his bed either by stretching their limbs or cutting them off) . Nevertheless, A. Einstein advises: Make everything as simple as possible, but not simpler. II’ll try my best to satisfy both. I let you know.Amtié. with the high r. A. Einstein: Make everything as simple as possible, but not simpler.Obviously a varicose vein isn't per se a disease but just a sign of venous system impairment responsible for a flow/pressure overload. Most of the time, this overload is due to superficial venous valve incompetence, but may be AV fistula, deep veins obstruction and congenital venous malformation. Duplex is able to assess all the etiologies. When the cause is sapheno-femoral valve incompetence and the hemodynamic feature is shunt I + II (frequent opportunity) hemodynamics guided mini-invasive surgery is safe and achieves a collapse of varices without any ablation and low recurrence rate attested by RC.So, particularly in young people, treating the varicose without ablation is feasible thanks to a proper knowledge of venous hemodynamics and a comprehensive Duplex assessment. See a video example: http://www.dailymotion.com/video/x5q55p\_chiva-shunt-i-et-ii\_techLaciste di Baker è un estenzione extra articolare della sinovia del ginocchio che puo anche estendersi entre l'adventice e la media dell'arteria poplitea. Puo essere causa di stenosi arteriosa poplitea o diThe hypothesis of patho-physiological correlation between chronic cerebrospinal venous insufficiency and multiple sclerosis: rationale of treatment  1. [M Lugli](http://phleb.rsmjournals.com/search?author1=M+Lugli&sortspec=date&submit=Submit)[⇓](http://phleb.rsmjournals.com/content/27/suppl_1/178.abstract#corresp-1)**,** 2. [M Morelli](http://phleb.rsmjournals.com/search?author1=M+Morelli&sortspec=date&submit=Submit)**,** 3. [S Guerzoni](http://phleb.rsmjournals.com/search?author1=S+Guerzoni&sortspec=date&submit=Submit)**and** 4. [O Maleti](http://phleb.rsmjournals.com/search?author1=O+Maleti&sortspec=date&submit=Submit)   [+](http://phleb.rsmjournals.com/content/27/suppl_1/178.abstract)Author Affiliations   1. Department of Cardiothoracic and Vascular Surgery, Hesperia Hospital, Via Arquà, 80, 41100 Modena, Italy 2. Correspondence: **Marzia Lugli MD** , Department of Cardiothoracic and Vascular Surgery, Hesperia Hospital, Via Arquà, 80, 41100 Modena, Italy. Email: [lugli@chirurgiavascolaremodena.it](mailto:lugli@chirurgiavascolaremodena.it)  Abstract **Background** The possible role of the venous system in the pathogenesis of chronic neurodegenerative diseases has been hypothesized for decades. Quite recently, the description of a venous condition defined as chronic cerebrospinal venous insufficiency (CCSVI) and its strong association with multiple sclerosis (MS) has brought back the attention of the scientific community to the hypothesis of an aetiological or concomitant role of an altered venous function in the occurrence of this pathology. CCSVI is identified by sonographic criteria, thus the indication for its possible treatment is based on ultrasound findings.  **Method** We retrospectively examined 167 consecutive patients affected by clinically defined MS and CCSVI, identified by ultrasound assessment by the presence of at least two sonographic criteria. Ultrasonographic diagnosis of CCSVI was then integrated by venography and intravascular ultrasound examination (in 43 patients). Patients were all submitted to endovascular procedure (venoplasty).  **Results** In 37% of cases there was no correspondence between the preoperative ultrasound assessment and the venographic findings. In the event of incongruity between venography and sonography, the intravascular ultrasound examination investigation, when performed, confirmed ultrasound findings in 42% of cases and venography results in 58%. At one month in 12% of cases ultrasound assessment showed the persistence of altered flux. In 67% of cases patients reported subjective amelioration, regarding non-specific symptoms.  **Conclusion** The pathophysiology of CCSVI is yet to be defined. The superior cava venous system is highly complex in terms of anatomy and possible anomalies, as well as its haemodynamic mechanisms. Further studies are required to define the parameters of diagnosis and treatment of CCSVI.  Keywords   * [chronic cerebro-spinal venous insufficiency](http://phleb.rsmjournals.com/search?fulltext=chronic+cerebro-spinal+venous+insufficiency&sortspec=date&submit=Submit&andorexactfulltext=phrase)      * [CCSVI](http://phleb.rsmjournals.com/search?fulltext=CCSVI&sortspec=date&submit=Submit&andorexactfulltext=phrase)      * [multiple sclerosis](http://phleb.rsmjournals.com/search?fulltext=multiple+sclerosis&sortspec=date&submit=Submit&andorexactfulltext=phrase)      * [IVUS](http://phleb.rsmjournals.com/search?fulltext=IVUS&sortspec=date&submit=Submit&andorexactfulltext=phrase) * [jugular vein](http://phleb.rsmjournals.com/search?fulltext=jugular+vein&sortspec=date&submit=Submit&andorexactfulltext=phrase)      * [azygos vein](http://phleb.rsmjournals.com/search?fulltext=azygos+vein&sortspec=date&submit=Submit&andorexactfulltext=phrase)      * [venoplasty](http://phleb.rsmjournals.com/search?fulltext=venoplasty&sortspec=date&submit=Submit&andorexactfulltext=phrase)   Dear Dr Eaton,  I’m sure of you sincerity.  The RCT I previously cited demonstrate that the functional and aesthetic results are better when the saphenous trunk is “hemodynamically” preserved are in contrast with your statement.  Others studies I previously cited attest that saphenous trunks is eligible for by-pass particularly when diameter is > or = 8 mm ( at the mid-thigh) and we use them particularly in the lower limbs for below-knee by-pass where the venous graft is still the best material for all the authors.  Internal mammary artery or a radial artery are not sufficient in case of multiple coronary by-passes ( see around you…)  Stenting is like here like in USA, generously used…and surgery takes place in case of stenting failure or not feasible, according to update literature.  Anyway, the informed consent must refer to RCT and studies of high quality and not to the individual opinion.  I’m am sure when you will try to perform CHIVA according to the hemodynamic laws and mini-invasive surgical technique as explicated in my book that I cited in my previous mail, your personal opinion will change…in accordance with my experience with so many other colleagues.  All the best  Most of the patients that we see have long standing truncal insufficiency with aneurysmal dilations and prolonged reflux. I have destroyed them without guilt. Previously I attempted to restore normal flow in the trunks but, even when successful, I found that success was short lived. Am I wrong to destroy those vessels that cause so much misery and degradation of quality of life?   Can you use those veins for coronary artery by-pass or other surgery?   Would you not prefer the left internal mammary artery or a radial artery for grafting? What is the current ratio of stent placement versus grafting surgery in France or Italy?   I look forward to your reply. Thank you.   Tom  Dear Dr Eaton and Alessandro  I send you privately my response because I sent it 3 das ago…and the moderator didn’t publish it.  I feel honored by your interest for CHIVA philosophy. Some exhaustive stripping+phlebectomy may give tremendous outcomes in ulcers....particularly when the draining perforator escaped fortunately from the surgeon. Recurrence after CHIVA? Sure, but not as much as the other methods, and easy to treat by the same method. Anyway, RCT and statistics exist because we cannot rationally generalize from 1 case. On another hand, drainage respect is also prevention from varicose recurrence as RCT proved it. In addition, preservation of the venous grafting capital is crucial, according not only to the studies but to my practice in a cardio-vascular surgery department and to my ethics concern. That information should be ethically and legally added to the informed consent of the patient. Some (several?) Italian doctors doesn’t perform CHIVA properly? Because too difficult or because they didn't learn it properly? Is medicine not reliable because of some bad doctors? Does patients deserve an effort of knowledge from their Doctor to give them the “best” treatment?  Principles of Venous hemodynamics by C.Franceschi and P.Zamboni 2010 ( Nova Biomedical www.novapublishers.com) could help who is eager to get an exhaustive information about CHIVA. On an other hand, I teach hemodynamics in French and Italian Universities.   Dear Dr. Franceschi,  Saphenous drainage is not always possible to restore and probably does not play as significant role as traditionally believed. I base this on having ablated the entire saphenous network (primary, secondary and tertiary vessels) in advanced cases where nearly all saphenous veins were dilated and insufficient. These patients had tremendous resolution of symptoms as well as resolution of prominent trophic changes, and in the ulcer patient, rapid and sustained ulcer healing. I do, however, remain impressed with the CHIVA philosophy.  Alessandro Frullini • Dear Claude  I know I am going to raise a problematic topic, and I agree RCT has shown some kind of evidence but the reality is that, al least in Italy where several doctors are performing CHIVA, we see so many patients with bad outcome after CHIVA. So the conclusion can be that 1) Chiva is so difficult to be properly performed and this make unrealistic its adoption or 2) for some reason that RCT are unrelaible ( who control the controler?).  Please don't consider this observation as simple criticism, the idea of haemodinamic control of venous insufficiency is great but we must face reality and, at least in this country, where Chiva is not so uncommon (even if is done in the absolute minority of patients) we face so often bad outcomes. Why we never heard of such patients in those studies? This morning I have seen a 42 years old patients who was treated with chiva on both legs years ago. The right one has now "only" a very large refluxing GSV >13mm but the left leg is in very poor condition with a larger GSV, venous ulcer, diffuse pigmentation, edema and several veins at risk of bleeding.  I believe that every treatment has failure in terms of bad control of the disease..... surgery, foam, thermal ablation (notwithstanding what Mark says) and obviously Chiva.  Ciao.  ,  The lack of venous capital secondary to varicose ablative treatment in patients who needed crucially an arterial by-pass induced me, 25 years ago1,2, to try to find an alternative method that could at the same time preserve the GSV and treat the varicose. The venous hemodynamic patterns were at that time, rather raw due to the limits of the investigation means. The advent of Ultra Sound Duplex scan provided an invaluable tool to investigate the hemodynamic “secrets” of the venous system. Thanks to this revolutionary technology, I could elicit not only the static data that matched with the previous studies but I could improve the understanding and assessment of venous dysfunction. Fortunately, these findings permitted me to elaborate therapeutic strategies and tactics that could achieve this double purpose. Further multicentric studies and long term RCT regarding Conservative and Hemodynamic treatment of venous Insufficiency in Ambulatory patients (CHIVA is the French acronym for Cure Conservatrice et Hémodynamique de l’Insuffisance Veineuse en Ambulatoire) showed the pertinence of these assumptions so that the no respect of the venous capital in the treatment of varicose veins is no more justified3,4,5,6,.  Dear Claude,  Thank you for your thoughts. It is discussions such as this that make me glad we have a forum like this, as it really comes to the heart of the philosopy of what we ar trying to achieve.  Firstly, as I said before, I would love to learn more about the understanding about CHIVA. However, in answer to your question, that is not really answerable. Thermoablation is a technique for destroying any vein that can be cannulated - CHIVA (from my understanding) is a haemodynamic approach to treating venous reflux. Hence one cannot be compared directly with the other - and presumably Thermoablation could be used in cerating segments of a CHIVA type operation?  As for the other points you make, they all come down to what philosophical approach that we have to the treatment of our patients.  My approach is to cure the veins as vertinly as possible with a minimal chance of recurrence in the future - thus ablate all reflux. In doing this I do not consider the future need for bypass, as few people will have all of thier veins ablated (meaning some will still have normal veins for a bypass), few people actually go on to need bypass surgery in thier lives, and for cardiac bypass, radial artery and internal mammary artery is optimal in many meaning only small amounts of vein (if any) will be needed.  Thus my feeling is that I would be doing what is in my view a sub-optimal opreration on many, which my well lead to further deterioration, so that a very few might benefit from having a vein that might be useful for bypass.  However I do understand and respect your view that this is the wrong way around and that we might consider preservation of the refluxing trucal vein for bypass.  Although I am always open to new thoughts and to reconsidering my practice and views, currenlty my patients want to get rid of their venous reflux and the sequelae of these, with the lowest possible chance of recurrence and best medical and cosmetic results.  As such, the elimination of reflux by thermaobaltion, coils, foam (if vein < 3mm), phlebectomy, TRLOP and microsclerotherapy are our optimal toolks at present which gives us a huge patient satisfaction and minimal recurrence rate (3.3% per annum and all of these are de novo reflux).  No RCT can be reported, as we haven't done one and no-one else uses our protocol. Similarly, I cannot see how I can do an RCT when my patients want the resutls that we are seen to obtain with our protocol.  I am very happy to discuss what we should be aiming for - but currently we are providing exactly what the patient asks for - the best possible results for their venous reflux problem with the longest lasting results.  Posted by Mark WhiteleyP.Zamboni and al Minimal Invasive Surgical Management of Primary Venous Ulcer vs. Compression Treatment: a randomized Clinical Trial  Eur J Vasc Endovasc Surg °°,1-6 (2003)  A mean follow up of 3 years  Conclusion: Hemodynamic correction ( the so called CHIVA treatment) eliminates reflux , while maintaining a saphenous drainage and leads to prolonged healing of venous ulcers in patients with primary isolated superficial reflux  Dear Mark,  Do you mean that Thermoablation is superior to CHIVA? Could you please let me know some reference? I gave mine.  Is it so clear to you that saphenous trunk ablation is better than conservative CHIVA procedure in terms of clinical outcomes? Reference? I gave mine.  If you state immoral to do a randomized study against stripping, does your moral concern extend to procedures that destroy the venous capital possibly vital in case of further needed arterial by-pass (1)? (despite EV procedures, venous grafting is still performed and saphenous trunks ( in varicose patients) are eligible for by-pass up to 8 mm diameter (2,3,4)).  Is it scientific to destroy the saphenous trunk when the EBM demonstrates (1A grade) the conservation superior to ablation?  Is it moral (and to-day legal) before destroying a saphena, not to inform the patient of the loss of opportunity for a further arterial by-pass? Particularly for a minor disease?. So let’s let him choose ablation, conservative procedure…or keep their varices and spider veins wearing support stockings.  1-Lofgren EP. In Bergan JJ, Lofgren EP. In Bergan JJ, Yao JST (eds). Surgery of the veins 1985 285-299 Cohn et al, Ann Thor Surg 2006 81(4) 1269-4  2-Wrapped autologous greater saphenous vein bypass for severe limb ischemia in patients with varicose veins. Preliminary report. [J Cardiovasc Surg (Torino).1995] PMID:7790328  3-Residual varicose veins below the knee after varicose vein surgery are not related to incompetent perforating veins. [J Vasc Surg. 2006] PMID:17098541  4-[Surgical techniques used for the treatment of varicose veins: survey of practice in France] [J Mal Vasc. 2003] PMID:14978433  5-Necessity of reconciling the objectives of the treatment of varices and arterial surgery. Practical consequences] [J Mal Vasc. 1991] PMID:1861112 [J Vasc Surg. 2006] PMID:16950441  Since CHIVA vs Stripping demonstrates RCT  new results that can be obtained by Thermoablation v Stripping.  our results are clearly superior to stripping - and we have presented this.  Horton was blasted by many professors of surgery showing that when something is so clearly superior to another technique, an RCT is not only not needed, but is morally wrong.  If it would be immoral to do a randomised study against stripping as we would be consigning half the patients to sub-standard outcomes.  Dear Professor Camilli  It is very, very elegant statement along Dr. Recek's Hemodynamic Paradox.   A few questions;  1. Could you explain more on your statement " popliteal-femoro-iliac may depend on many factors, at supra- and infra-inguinal level, effective in about 30-40% of patients with relapsing VVs) " as below?   2. It would be nice to explain on the fact " The CHIVA technique (conservative) showed late results better -------, this may depend on reservoir volume reduction (decongestion) more than on pressure reduction". Your interpretation?  And I also wish Dr. Recek to share his interpretation on Prof. Camilli's statement.  Regards,  BB Lee, MD  From: [vasculab@yahoogroups.com](mailto:vasculab%40yahoogroups.com) [mailto:[vasculab@yahoogroups.com](mailto:vasculab%40yahoogroups.com)] On Behalf Of sante camilli Sent: Sunday, March 11, 2012 4:41 AM To: Vasculab  Subject: RE: [vasculab] Hemodynamic paradox  Dear Colleagues Recek and Franceschi and Simka and Lee and All,  about the â hemodynamic paradox â (proposed by Recek) and the brilliant refinement with â electrical circuits Ã¢â‚¬diagram (proposed by  Simka), I would add some comments. A.  The Ã¢â‚¬Å“hemodynamic paradoxÃ¢â‚¬Â is a real thing but it works variably and in not predictive way, depending on many components in individuals (genetical and acquired factors, residual escape points, shunt resistance, reservoir volume, time, others). These components aren't easy to detect and analyze separately, but if we were able to fight against them we may delay the varicose recurrence appearance and slow down their worsening. B. Since varicose veins (VVs) dilation relates to â venous load â more than to â ambulatory venous pressure â, we need to know better the venous load behavior. Unfortunately it's difficult to check and measure during activity.  C. In my experience and opinion, the venous load depends on many and inter-related factors (a real complex and variable web!): standing position (which generates gravitational force); hydrostatic pressure gradient (depending on the length of incompetent venous segment); superficial and deep system resistances (e.g. popliteal-femoro-iliac may depend on many factors, at supra- and infra-inguinal level, effective in about 30-40% of patients with relapsing VVs); shunt circuit capacity (reservoir volume); it may grow largely on the superficial system, meanwhile on the deep system its enlargement is barred by muscles and fascia; reflux speed and amount (depending on gradient (b), shunt resistance, incompetent venous cross-sectional area, escape points, reservoir volume (d), others) muscular activity and pumping, which generates energy against the gravitational force (a) and pressure gradient (b), and a consequent overflow, overload, overshear stress, etc. (as a relation with c+d+e+g); time; others.  All these factors are involved in VVs recurrence, but in different manner; this is the topic we need to study.   D.  At a time done, among these factors, the reservoir volume (d) is probably the most effective. In fact, a+b+c+f+g are mostly not modifiable factors. The only really modifiable factor is (d) (by phlebectomies, CHIVA drainage, sclerotherapy, bandages, stockings, phlebotonic drugs, Ã¢â‚¬Â¦), meanwhile (e) may diminish according to diminution of (d). The overload (physiologic + shunt load) depends on (d) x (energy); the energy being due to pressure gradient (b) (activating the backward flow, the stable factor) and to the volume (d) x muscle activity/pumping (f) (activating the forward flow speed, the extremely variable factor). So, the reservoir volume (d) intervenes in the expression of multiple factors.  E. The CHIVA technique (conservative) showed late results better than those of stripping (ablative). Hypothesis: this may depend on reservoir volume reduction (decongestion) more than on pressure reduction.   I would have very grateful to all of you for any comments and also for any suggestions regarding an experimental model or study-design focused on the topic.   All the best.  Prof. Sante Camilli M.D.Consultant in Vascular SurgeryVia Lombardia 30 - 00187 Roma, Italiae-mail: [sacami@hotmail.com](mailto:sacami%40hotmail.com)<mailto:[sacami%40hotmail.com](mailto:sacami%2540hotmail.com)> , [sante.camilli@gmail.com](mailto:sante.camilli%40gmail.com) <mailto:[sante.camilli%40gmail.com](mailto:sante.camilli%2540gmail.com)>  \_\_.\_,\_.\_\_\_  I agree with 90% of Sante model which matches with the haemodynamic basis I designed for CHIVA.  Nevertheless, I don’t fully agree with his haemodynamic explanation for the better late CHIVA results vs Stripping, where he suggests “this may depend on reservoir volume reduction (decongestion) more than on pressure reduction” because this assumption doesn’t fit to physics laws nor to my experience. Reservoir effect being the venous system capability to vary little its inner pressure when submitted to greater pressure/volume supplement, by increasing its volume thanks to its wall Compliance. As a matter of fact, this phenomenon is mostly passive and caliber reduction after CHIVA is due to Pressure//Volume overload ablation (walking Closed shunts pressure/volume and standing Gravitational pressure) while the veins remain conserved in order to drain the "physiologic flow" whatever the direction ( down or upwards) provided it respects the drainage hierarchy from surface to depth, as a prevention for skin suffering and vicarious (by-passing) recurrent varices.  Dr. Camilli touched on the interesting topic about circumstances that could affect formation of recurrences, and mentioned a few terms, such as *venous load, overload, overflow, shunt resistance, superficial and deep system resistance*, and some others. Several questions arise in that connection: How can these circumstances be measured? In which units quantified? How do they influence, if at all, the probability to recur? If so, in which direction? Which of them retard and which accelerate recurrences? I am afraid, some of these questions will remain unanswered for a long time.  C. Recek  Dera Fausto  I agree with majority of your comments.  However, you are discussing the situation of a subject who is standing still, and a reflux is provoked either with manual compression, or with Valsalva, Parana, etc. (in general, a hemodynamic pattern found during sonographic assessment of the leg, with already filled incompetent superficial veins).  And now - imagine a subject who is changing his body position from supine to standing, or - he is walking, raising the leg, etc. I wonder if your statements about minimal relevance of superficial network are correct. Perhaps you are right, but actually, we simply DO NOT know.  Unfortunatelly, most of the researchers are fascinated by things like lasers, and no longer perform boring studies on venous hemodynamics. Consequently, we are doomed to treat our patients according to conjectures, and not according to the evidence, which is not a good thing.  Any thoughts  Marian Simka  Dear Dr Recek, BBLee and all,  About misunderstanding about vascular specialists: Causes for misunderstanding are several.  a- Discordant expertise in theoretical ( and practical) venous hemodynamics where sufficient culture in physics and US Duplex practice is mandatory.  b- Misused terminology when the related concepts and definition are not clearly known (understood) or expressed. For my own, I described and explained exhaustively the new (or renewed) concepts since 20 years and at least in an American Book ( Principles of venous hemodynamics: Novapiblishers.com). So any new concept need a new name (is any new name jargon?) and any name must represent a concept clearly defined.  c- Discordant appraisal of evidence. Many specialists are trusting in venography, plethyspmography, invasive pressure measurements but they underestimate Duplex Scan capabilities because they underuse or misunderstood them. So For decades I works in US engineering and in Doppler signal semiology since decades ( I wrote the first book in the world about Doppler signal semiology resulting from my research ( L’Investigation Vasculaire par Ultrasonographie Doppler Masson Edit 1977 translated in Spanish and Italian) , despised for decades…and nowadays worldwide accepted.  d- Evidences in Venous US Duplex Scan are provided by anatomical vision of the venous network and flow assessments at rest , in various postures and valvo-muscular pump activation (e.g Parana manoeuver). Since the direction and flow velocity measurement by US is reliable, data such as velocity and flow direction in the veins are per se evidences. Then the interpretation of these data according various physiological and pathophysiological conditions and fluid mechanics laws, depends on the underlying hemodynamic model (e.g vicarious shunts, closed shunts, dynamic fractioning of hydrostatic pressure etc…) previously in accordance with physics. These are evidences not only in terms of their inter practitioner reproducibility but in terms of predictable outcomes after treatments in accordance with the underlying hemodynamic model , demonstrated for CHIVA by long term RCT follow-up.  e- Conclusion: I suggest my sceptic colleagues to study deeper the venous hemodynamics and US duplex Scan, particularly those related to CHIVA. Those who did it are convinced and enthusiastic with their hemodynamic and conservative treatments.  *Dear BBLee,*  *I’m back to France. So I can reply to:”But the sticky point here in this new approach is, when this perforators loses its mandated function as one way escape route due to its own valvular dysfunction, then, how much we gain by the preservation of such*  *malfunctioning/crowded/two way communicating escape routes?”*  *This opinion which still command to destroy all the perforators (large and small) because supposed to be responsible for present and potential varicose veins is contradicted by the facts and evidences : Dr RECEK demonstrated that below knee perforators are not refluxing most of the time even if they are large ( we could call it "perforator paradox") and Doppler assessment confirms this every day. On the other hand, CHIVA vs Stripping follow up ( 5,10 years) didn't show recurrence due to below knee incompetent perforators in CHIVA group nor in stripping group. Their size is not proportional to the reflux but to the inward re-entring overloaded flow by upper escape points.*  *( about Doppler venous pressure measurement: I always refer to my passed friend Mauro Bartolo and I made a video tribute some years ago ).*:  Dear Dr. Recek  Very interesting observation!  Nevertheless, are these two spikes(?)/upsurge of the flow along the saph vein on the graph in the initiating period of the calf muscle contraction/pump reflecting delayed valve closure?    Here in the U.S., we define the outward flow (flow direction from the perforator vein to the superficial vein) of > 350 ms as reflux in general since most of  normal limbs shows the outward flow is < 350 ms and most normal perforator veins have a subfascial diameter of< 3 mm. Based on the data along other veins, most of centers seem to use a duration > 0.5 s as a cutoff for perforator reflux. Although the larger diameter indicates the presence of reflux with high certainty, one-third of refluxing perforator veins have a smaller diameter than these cutoff points.    1.      Nevertheless, most of the literatures we use as a reference for the perforators  quotes subfascial bicuspid 'unidirectional' valve as the rule allowing the blood to flow from superficial veins to the deep veins (inward flow) except the perforators in the foot- I do NOT mean the communicating vein-.  2.      Only literature in my reference files- not updated for more than one year now!- to mention on this 'bidirectional' flow in perforator veins in the calf is Pirner F. Die Bedeutung der insuff. V. perforans fur die Krampfadeoperation. Chir Prax. 1963;7:112-119.    Any comment?  BB    **From:** vasculab@yahoogroups.com [mailto:vasculab@yahoogroups.com] **On Behalf Of**Cestmir Recek **Sent:** Friday, March 30, 2012 2:22 PM **To:** vasculab@yahoogroups.com **Subject:** [vasculab] Calf perforators      Dear all,  Invasive recordings of the factual pressure in the GSV in healthy persons could deliver the conclusive proof that lower leg perforators as a system are competent, that they do not allow escaping of venous blood from deep into superficial veins. In such a case, the pressure curve should descend stepwise during calf pump activity, without upwards peaks, as schematically shown on the attached picture. Interestingly, on the figure by Höjensgard and Stürup I presented in a previous message such a situation**exceptionally** happened (tagged), whatever the reason might be (see attachment). Otherwise the figure by Höjensgard and Stürup delivered a proof confirming bidirectional flow.  Disagreeable for the advocates of the competence theory: pressure measurements performed as yet in healthy persons in the GSV by Höjensgard and Stürup and Arnoldi showed distinct increase in pressure during calf muscle contractions.  The possibilities of DUS to evidence that lower leg perforators as a system hinder escaping of venous blood from deep into superficial veins are limited. On the one hand, DUS examines particular perforators, not the whole system. There is allegedly a lot of perforators beneath the knee. On the other hand, even if DUS detects inward flow but no outward flow in a calf perforator, that cannot be regarded as a conclusive proof excluding bidirectional flow. The inward flow is larger, and therefore easier to detect than the weaker outward flow; may be it exceeds the capability of DUS to detect a weak outward flow in slight channels within a fraction of a second.  C. Recek  Swell Dr Recek  The  invasive pressure measurement you are  refering  to  matches with our DUS findings when we see a reflux at the calf perforators during the contraction ( systole),  but much smaller than the inward flow during the relaxation (diastole). We consider this "normal". BUT this systolic reflux is not seen everytime nor in every calf (leg) perforator . Your hypothesis is that DUS can't "see" a too fast reflux ( fraction of second) is possible but not likely with the today technology.  An other hypothesis could be that a brutal stop of flow gives a static pressure rebound ( Bernouilli) and or an outward instant pressure wave transmision during the perforator valve closure and/or a slight telesystolic reflux accompaining these valves closure comparable with what is seen during the diastolic closure of the deep and superficial veins.  Thank for the oxygen you blow into Vasculab.  Dr Claude Francesch  Dear Claude  So you think an outward instant pressure wave transmision during the perforator valve closure.  Very fascinating interpretation based on  Bernouilli's law!  But what do you mean by 'a slight telesystolic reflux accompaining these valves closure'?  Regards,  BB  P.S. As I inquired to Dr. Recek, the cutoff time for this perforator reflux seems to be very crucial. As far as I know, most agrees with the outward flow of > 350 ms as reflux based on the normal limb findings although the vast majority shows even lower value (< 200 ms). But I believe most colleagues use a duration > 0.5 s as a cutoff for perforator reflux since this outward flow of 350 ms duration is so short, and also for other veins  a cutoff value of > 0.5 s is common (with the exception of common femoral, femoral, and popliteal veins  with > 1 s). But I also think the size/diameter of perforator veins in a subfascial level should be considered as a major factor to affect on this reflux/valvular insufficiency. Nevertheless, one-third of refluxing perforator veins have a smaller diameter than these cutoff points although the larger diameter indicates the presence of reflux with high certainty.  .  An other hypothesis could be that a brutal stop of flow gives a static pressure rebound ( Bernouilli) and or an outward instant pressure wave transmision during the perforator valve closure and/or a slight telesystolic reflux accompaining these valves closure comparable with what is seen during the diastolic closure of the deep and superficial veins.  Dear Professor BB Lee and Dr Recek,    I believe we see this every day as antegrade flow within the saphenous vein on calf compression or tip-toe.  This flow "must" come from reflux/outward flow in perforating veins.    Best wishes,  There we go, Chris. Glad you also read this chat room talks. Welcome aboard.  Since the revision of CEAP classification has been so bitterly blamed on the ignorance to reflect European colleagues' opinion properly -issue on the telangiectasia, etc-, I want to make sure we don't make same mistake and want to make sure not to miss even 'unofficial' casual opinion to be reflected properly in current IUP Consensus you are also involved.  And this vasculab communication provides quite a substantial information on various backgrounds on the Venous pressures, flow, velocity, resistance, wall compliance and the direction, etc.  So this communication Dr. Recek initiated will certainly be reflected as a part of our hemodynamic consensus (Section 3. Physiology of Venous Circulation) as far as there is a solid evidence-based data supporting these rich speculations.   Regards, BB  Dear Massimo  I enjoyed your re-interpretation of Claudio's statement based on the energy equation (Energy = Pressure x Volume).  But, Claudio's statement: "The greater the velocity, the less the lateral pressure on the wall" is also correct one based on classical Bernoulli's equation (static pressure + dynamic pressure = total pressure) we are all familiar with.   It looks like everyone seems to enjoy a refreshing course of the fluid-dynamics involved to this reflux issue.  But nobody seems to care about the fact that Bernoulli’s equation is imperfect because it overlooks the viscosity of fluids (η), and Poiseuille’s equation as well is imperfect because it neglects gravitational pressure and gravitational potential as well as accelerative-decelerative pressures.  Further, nobody raised an issue on this Bernoulli’s equation which was formulated for the flow that is steady and not pulsatile, and laminar/streamline moving in multiple layers within the 'incompressible' tube.   Indeed, not all the readers would know in depth on the fact that such physics laws like Bernoulli's or Poiseuille’s equation are not fool-proof for the circulation system. So, I worry about how ordinary clinicians would interpret your illustration solely based on the Bernoulli's equation/law. And you did not mention a single word all along about the Bernoulli-Poiseuille equation as a combination of the two as the more realistic equation.  Any comment?  BB  Dear BBLEE and Chris Lattimer,  A/About telesystolic valve closure  The “normal” outflow described by Dr Recek is proto-systolic (due to an outward pressure gradient triggered by calf contraction) could be due ( hypothesis) to the same reason why the proto-diastolic back flow occurs in normal deep and superficial veins (due to a reverse pressure gradient triggered by calf contraction relaxation).  B/ About GSV flow feeding  GSV flow is permanently fed by the draining microcirculation of the skin . An additional flow occurs during calf contraction and tip-toe.  Just test your assumption: watch the GSV flow:  1/ during raising on tip-toe ( or Paranà manoeuver) with and without a tourniquet tied at the ankle  2/ On squeezing the foot  3/ On squeezing the calf  Perhaps, you will observe that this additional flow is due :  1/ During raising on tip-toe to the plantar venous pump emptying ( lejars pump , the only place where perforators are “normally” incompetent and refluxing)  2/ and when squeezing to the direct manual compression of the GSV.  During raising on tip-toe this systolic flow appears in the GSV despite the tourniquet , a systolic reflux will be found into one or more calf perforators. This occurs frequently in refluxing GSV and precedes a much more important inward diastolic flow. This may be due to the incompetence of the perforator valve when dilate (preferential route) despite perfectly in ward re-entering diastolic flow ) . Otherwise, may be due to a deep vein block ( permanent or only dynamic ).  C/About fluid mechanics and venous hemodynamics:  Fluid dynamics complexity is due to the non-linearity of the parameters which values and weight change dramatically with the initial data , velocity, caliber , compliance, gradient pressure,etc…as summarized in Navier-Stokes equations. Nevertheless simplified equations are sufficient to make the aircrafts fly. The same simplification is implemented in cardiac and arterial hemodynamics...and it works. Simplification is also unavoidable and pertinent in venous hemodynamics. I.e, Reynolds number is predominant only when the velocity is very high and refers to turbulences, Poiseuille equation is applicable when the caliber is very small and refers to resistances to the flow, Bernouilli equation is useful when the velocity variation changes the static energy in dynamic energy and vice versa . Physics is necessarily an approximation of the reality. ( see lower equations copied and pasted from Wikipedia).  **Bernoulli's principle** states that for an [inviscid flow](http://en.wikipedia.org/wiki/Inviscid_flow), an increase in the speed of the fluid occurs simultaneously with a decrease in [pressure](http://en.wikipedia.org/wiki/Pressure) or a decrease in the [fluid](http://en.wikipedia.org/wiki/Fluid)'s [potential energy](http://en.wikipedia.org/wiki/Potential_energy).  \tfrac12\, \rho\, v^2\, +\, \rho\, g\, z\, +\, p\, =\, \text{constant}\,  v\, is the fluid flow [speed](http://en.wikipedia.org/wiki/Speed) at a point on a streamline,  g\, is the [acceleration due to gravity](http://en.wikipedia.org/wiki/Earth%27s_gravity),  z\, is the [elevation](http://en.wikipedia.org/wiki/Elevation) of the point above a reference plane, with the positive *z*-direction pointing upward – so in the direction opposite to the gravitational acceleration,  p\, is the [pressure](http://en.wikipedia.org/wiki/Pressure) at the chosen point, and   * is the [density](http://en.wikipedia.org/wiki/Density) of the fluid at all points in the fluid   In [fluid dynamics](http://en.wikipedia.org/wiki/Fluid_dynamics), the **Hagen–Poiseuille equation** is a [physical law](http://en.wikipedia.org/wiki/Physical_law) that gives the [pressure](http://en.wikipedia.org/wiki/Pressure) drop in a fluid flowing through a long cylindrical pipe. The assumptions of the equation are that the flow is laminar [viscous](http://en.wikipedia.org/wiki/Viscous) and[incompressible](http://en.wikipedia.org/wiki/Incompressible) and the flow is through a constant circular cross-section that is substantially longer than its diameter  **Standard fluid dynamics notation**  In standard fluid dynamics notation:[[1]](http://en.wikipedia.org/wiki/Hagen%E2%80%93Poiseuille_equation#cite_note-Kirby-0)[[2]](http://en.wikipedia.org/wiki/Hagen%E2%80%93Poiseuille_equation#cite_note-Bruus-1)  \Delta P = \frac{8 \mu L Q}{ \pi r^4}  where:  \Delta P  is the pressure drop  L is the length of pipe  \mu  is the [dynamic viscosity](http://en.wikipedia.org/wiki/Dynamic_viscosity)  Q is the [volumetric flow rate](http://en.wikipedia.org/wiki/Volumetric_flow_rate)  r is the [radius](http://en.wikipedia.org/wiki/Radius)  d is the [diameter](http://en.wikipedia.org/wiki/Diameter)  [Description :  \pi](http://en.wikipedia.org/wiki/Pi)  is the mathematical constant  Reynolds number  The assumption of inviscid flow is generally valid where [viscous](http://en.wikipedia.org/wiki/Viscosity) forces are small in comparison to inertial forces. Such flow situations can be identified as flows with a [Reynolds number](http://en.wikipedia.org/wiki/Reynolds_number) much greater than one. The assumption that viscous forces are negligible can be used to simplify the [Navier-Stokes solution](http://en.wikipedia.org/wiki/Navier-Stokes_equations) to the [Euler equations](http://en.wikipedia.org/wiki/Euler_equations_(fluid_dynamics)).  While throughout much of a flow-field the effect of viscosity may be very small, a number of factors make the assumption of negligible viscosity invalid in many cases. Viscosity cannot be neglected near fluid boundaries because of the presence of a [boundary layer](http://en.wikipedia.org/wiki/Boundary_layer), which enhances the effect of even a small amount of [viscosity](http://en.wikipedia.org/wiki/Viscosity). [Turbulence](http://en.wikipedia.org/wiki/Turbulence) is also observed in some high-Reynolds-number flows, and is a process through which energy is transferred to increasingly small scales of motion until it is dissipated by viscosity.  **Navier–Stokes equations**, named after [Claude-Louis Navier](http://en.wikipedia.org/wiki/Claude-Louis_Navier) and [George Gabriel Stokes](http://en.wikipedia.org/wiki/Sir_George_Stokes,_1st_Baronet), describe the motion of [fluid](http://en.wikipedia.org/wiki/Fluid)substances. These equations arise from applying [Newton's second law](http://en.wikipedia.org/wiki/Newton%27s_second_law) to [fluid motion](http://en.wikipedia.org/wiki/Fluid_dynamics), together with the assumption that the fluid [stress](http://en.wikipedia.org/wiki/Stress_(physics))is the sum of a [diffusing](http://en.wikipedia.org/wiki/Diffusion) [viscous](http://en.wikipedia.org/wiki/Viscosity) term (proportional to the gradient of velocity), plus a [pressure](http://en.wikipedia.org/wiki/Pressure) term. |  |

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| **[1] Carandina S, Mari C, De Palma M, Marcellino MG,Cisno C, Legnaro A, et al.Varicose Vein Stripping vsHaemodynamic Correction (CHIVA): a long term randomised trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230–7**  **[2] Parés JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX,et l.Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624–31**  **[3] Iborra-Ortega E, Barjau-Urrea E, Vila-Coll R, Ballon-Carazas H, Cairols-Castellote MA. Comparative study oftwo surgical techniques in the treatment of varicose veins of the lower extremities: results after five years of followup. Estudio comparativo de dos técnicas quirúrgicas en el tratamiento de las varices de las extremidades inferiores: resultados tras cinco años de seguimiento]. Angiología 2006; 58(6):459–68.**  **[4]P.Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. Compression Eur J vasc Endovasc Surg 00,1 6 (2003)**  **[5] Bellmunt-Montoya S, Escribano JM, Dilme J, Martinez-Zapata MJ. CHIVA method for the treatment of chronic venous insufficiency. Cochrane Database of Systematic Reviews 2012 , Issue 2 . Art. No.: CD009648. DOI:10.1002/14651858.CD009648 .**  **[6] Chan, C.-Y.a , Chen, T.-C.b , Hsieh, Y.-K.a , Huang, J.-H.c  Retrospective comparison of clinical outcomes between endovenous laser and saphenous vein-sparing surgery for treatment of varicose veins (2011) World Journal of Surgery, 35 (7), pp. 1679-1686.**  **[7] Milone M, Salvatore G, Maietta P, Sosa Fernandez LM, Milone Recurrent varicose veins of the lower limbs after surgery. Role of surgical technique (stripping vs. CHIVA) and surgeon's experience.F. G Chir. 2011 Nov-Dec;32(11-12):460-3**  Sorry, Claude, but again I would like to verify what you described as " surprised by the NORMAL draining function of the below knee perforators, whatever their size. Very few of them are refluxing." Your as well as Dr. Recek's claim on new interpretation of the 'reflux' issue will remain as one of major sticky points provoking substantial confusion especially among the students to traditional concept/school like me and ALL my colleagues on this side of the Atlantic.  So we need more clear description on such basic/fundamental issue with sufficient example- in that regard, Stefano Ermini is the one who is most effective to deliver what he wants through the video-. Indeed, no matter how logical this new hemodynamic interpretation would be, the current hemodynamic principles Eugene Strandness and David Sumner established will remain the base for all the saphenous vein-related practice till proven otherwise!  We would need more efforts in this regard.  Gingerly,  BB |

Dear Claude,

The diagram is brilliant! Perfecto to summarize what you claim for; I couldn't agree more on such 'logically perfect' approach.

But, honestly, I wonder how many cases in daily practice would fall into this category/indication you have shown through the diagram.

BTW, I want to make sure I understand correct that without Step 2 for a-b (CS disconnection), Step 3 for c-e alone will NOT be sufficient, correct?-.

Further, we still honor the fundamental concept/skepticism on the manipulation of the deep vein system itself based on what we actually learned through decades, and have a fear to attack the deep system as the way you promote to restore the competency.

So, the removal/ligation of a part of the deep vein/posterior tibial vein itself, only to arrest the reflux/insufficiency while the combined condition of the obstruction remains, is quite intriguing.

Perhaps, only CHIVA students with top notch qualification on the DUS like you or Ermini could carry on with a good chance to success but I wonder how many are capable/competent to deal with such bold approach/manipulation of the deep vein especially below the knee with much 'limited' flow/volume through low pressure system, in order to tackle the reflux portion alone out of combined 'obstruction-reflux/insufficiency' status.

More I learn on this NEW(?) hemodynamic interpretation on the reflux issue, more I get mind boggling! And I don't know I am ever able to reach to the point to convince myself with a self-satisfaction.

All the best,

BB

Mon dieu!

You should read carefully my answer. The relationship between pressure and speed is present when the ENERGY EXIST!

If you stop the reflux the energy developed by the pump is not delivered to the system, that remain without motive energy, consequentially speed and static diastolic pressure disappears, only hydrostatic and residual pressure will be detectable. How can you image an inertia with liquids that are incompressible in a full system with low compliance ?

A resistance has different effects on pressure and speed depending on wether the pump is placed upstream or downstream.

When a resistance is placed downstream a pump it engages the distribution between pressure and speed. It reduces the speed and increases the pressure (first principle of the thermodynamic)

When a resistance is place upstream a pump (aspirative pump) it engages the DEGREE OF ENERGY DELIVERED TO the system with lowering of pressure and speed.

Chew on it!

Massimo Cappelli

Dear Massimo

Could you do me a favor? I know you are one of guru in the field of hemoynamics but unfortunately we are NOT all experts in the hemodynamics like you or Stefano. Majority of this chat room communicators are practical clinicians like me- hope I am wrong!-

It would be very nice of you to lower your eye level to our -rather my humble level- with diligent explanations on what " THE SPEED STOPS AND THE PRESSURE FALLS DOWN " like Hemodynamics-101 I taught to Biomedical Engineering Postgraduate students for more a decade.

I would personally appreciate more to you to share your vast hemodynamic knowledge with us on your statement " Other thing is the presence of an anterograde systolic flow, in this case the pressure rises when the anterograde flow is performed stopping the retrograde telediastolic back flow " with more substances.

Warm regards,

BB

I buy your points, Claude and trust your claim. But in a way this 'challenging' approach is almost revolutionary shaking the base of what we believe now- I am talking about PTS(post-thrombotic syndrome).

I think it should be worthy for your crusade(?) with the video if you could spare the time to edit and put it on the YouTube as Stefano does, including the diagram you posted on the last letter which certainly will give an extra dividend to your work.

All the best,

BB

P.S. I wish Fausto to make an independent communication for this issue like a systolic-diastolic reflux issue and invite further serious reviews on three separate conditions: obstruction-reflux combined condition, obstruction alone, and reflux alone like a primary/essential type of the insufficiency

Sorry to interfere, Claude, but could you add more explanation on your statement " --- relate to their closed angulation according to the deep flow direction --- doesn't overload that much the GSV except perhaps during intensive exercise , so explaining part of the large superficial veins no refluxing in diastole despite "varicose"  in sportsmen"?

Thanks,

BB

David

As far as I know of, no one among our camp(?) on this side of the Atlantic would dare/willing to cross the line to attack/ligate the deep vein per se but remain at the level of connector/perforators to allow the reflux and get out instead of looking for the deep vein to provide the source of leakage/reflux.

Perhaps due to the 'indiscriminating' attack even to these perforators, SEPS lost its initial lusts and I understand the review board recommended to the insurance companies to stop paying on the SEPS here in the U.S. which might be the right decision till we know more on this reflux business.

That is one of the reasons why I am intensely interested in this new hemodynamic interpretation- actually NOT new since Claude brought this concept in 1988 if I remember correct- in view of such unpredictable, mostly disappointing, outcomes of the SEPS as well as SSV ligation at its SPJ through the years. Indeed, I still remember that how much we were really amazed through first half dozen cases of SEPS to see so many perforators blocking(?) our way to reach to the culprit perforators beneath the ulcer base and they all looked same to confuse us to find a genuine culprit to cause an indolent ulcer.

I happen to belong to the old generation more comfortable with the ascending venography so that I still have some difficulty to swallow the DUS-based tibial vein resection only to remove the reflux issue as Claude does on the condition of obstruction-reflux combined status.

Regards,

BB

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Dear BB,  
Deep Chiva could be a good topic to discuss in an Event like we did for Systodiastolic.   
However, I will do it at the end of May, after my participation to Australasian ACP in March and my congress on tromboembolism in May.   
Wait for that.   
Regards  
Fausto

Thanks, John, for such extensive information on your work.

Of course, I am very familiar with your unique work with great admiration because I have been struggling with the condition of avalvulosis among truncular venous malformation through decades. Despite much efforts for more than two decades, I didn't have much luck but the frustration!  I even invited once Professor Victor Krylov of Moscow Institute of Advanced Medicine to my lab to learn/master Russian technique to create a genuine vein valve he created as a flap covered with sound endothelium on whole surface from the outset. But partly based on the practical/clinical implication difficulty besides the fund ran out, we abandoned after extensive peer review by my mentor, John Bergan.

So, when we did have quite substantial discussion back in 2008? through this Vasculab, I raised two specific questions to you, one for the endothelialization of the adventitia side of the cusp and also the fate of the suspension sutures/strings. I don't recall clearly but you mentioned that whole adventitial surface was eventually replaced/covered with the endothelium to warrant its longevity.

Since then, I inquired many colleagues who committed to this valve reconstruction on this rationale but all disagreed with your expectation. Even Prof. Krylov, the world pioneer of vein valve reconstruction flatly denied my wishful thinking based on his animal experiment when he visited me a few years ago. That is the reason why I raise same questions as I did back in 2008, and hoped any, among our own colleagues, was able to reproduce your admirable data since 2008.  Yes, I also had a chance to talk with Frank Veith last December to emphasize more support/interests among the colleagues especially on your as well as Maleti-Lugli's valve work while discussing on Dusan Pavcnik's life time dedication on the artificial valve creation.

It would be nice of you to respond me one more time on these TWO specific issues.

Respectfully,

BB

P.S. I take the liberty to forward this communication to Maleti and Lugli of Modena group to get their opinions if possible.

P.S. Some other technical issues on your work: how do you choose the site for anchoring the suspension string and how long(length)? And how do you tailor the shape of the cusp to fit perfect to circular(?) lumen with no leak? Do you clean irregular surface of the lumen with full of scars?

Stefano,

I hope European colleagues learn the lesson from what we went through the battle(?) between the APG and PPG more than two decades ago. Since APG got rid of PPG from the U.S. market despite vehement protests by a few who supported PPG over APG, all the earlier PPG works became dead because those PPG data were not able to compare with the APG data accepted as so called common language. And we are virtually trapped by this monopolizing APG which is too expensive. In my opinion, PPG, either arterial or venous, are a lot more economical and sensible for daily use with minimum expense.

BB

**Faudra-t-il prescrire une statine à tous les hommes de plus de 50 ans ?**

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| Publié le 29/05/2012   | [1 réaction [http://www.jim.fr/images/puce_reaction.png](http://www.jim.fr/e-docs/00/02/03/CF/index.phtml#reactions)](http://www.jim.fr/e-docs/00/02/03/CF/index.phtml#reactions) | [Partager sur Twitter](http://twitter.com/intent/tweet?text=Faudra-t-il+prescrire+une+statine+%C3%A0+tous+les+hommes+de+plus+de+50+ans+?++:+http://www.jim.fr/203CF) [Partager sur Facebook](http://www.facebook.com/sharer.php?u=http://www.jim.fr/e-docs/00/02/03/CF/document_actu_med.phtml) [Imprimer l'article](http://www.jim.fr/print/e-docs/00/02/03/CF/document_actu_med.phtml) [Envoyer à un confrère](http://www.jim.fr/mail/index.phtml?url=http://www.jim.fr/e-docs/00/02/03/CF/document_actu_med.phtml) [Réagir à l'article](http://www.jim.fr/mail/reagir.phtml?cle_doc=132047) [Enregistrer dans ma bibliothèque](http://www.jim.fr/e-docs/00/02/03/CF/enreg_biblio.phtml) [Reduire](javascript:selectStyleSheet(-2);;) [Agrandir](javascript:selectStyleSheet(2);;) |

Cette question qui aurait semblé hors de propos,  il y a quelques années, est pourtant celle que pose l’éditorialiste du très austère Lancet en commentant la nouvelle méta-analyse prospective des Cholesterol Treatment Trialists’ (CTT).

Ce groupe intègre depuis 1994 dans un travail de méta-analyse régulièrement mis à jour, toutes les données individuelles des patients inclus dans des essais randomisés en double aveugle portant sur les traitements par statines en prévention cardiovasculaire (CV). Il avait montré dès 2005, sur les 90 056 premiers sujets inclus, qu’une baisse de 1 mmol/L du taux de LDL cholestérol (0,39 g/L) sous statines réduisait en 5 ans la fréquence des infarctus du myocarde de 23 %, celle des événements vasculaires majeurs de 21 %, celle de la mortalité coronarienne de 19 % et la mortalité globale de 12 % (toutes ces diminutions étant hautement significatives ; p<0,0001). De plus ces baisses de la morbi-mortalité CV  avaient été constatées indépendamment de l’âge, du sexe, du type d’indication (prévention primaire ou secondaire), de la présence ou de l’absence d’un diabète ou d’une hypertension et surtout quel que soit le niveau de LDL-C de départ (y compris pour des taux inférieurs à 3,5 mmol/L).

**Un bénéfice significatif même pour les sujets à faible risque cardiovasculaire**

L’analyse actuelle des CTT porte sur 134 537 sujets inclus dans des essais randomisés statines contre placebo et 39 612 patients dans des essais comparant un traitement standard par statines à un traitement intensif (études SEARCH, A to Z, TNT, IDEAL et PROVE-IT) (1). L’objectif était cette fois de déterminer si l’effet sur la morbi-mortalité CV était corrélé au niveau de risque CV initial en comparant les résultats d’une baisse de 1 mmol/L de LDL-C pour 5 catégories de patients ayant une probabilité croissante d’événements vasculaires majeurs à 5 ans : inférieure à 5 %, entre 5 et 10 %, entre 10 et 20 %, entre 20 et 30 % et supérieure ou égale à 30 %. Il est apparu, qu’en valeur relative, le bénéfice des statines était au moins aussi important pour les 2 niveaux de risque les plus faibles que pour les plus élevés. Ainsi par exemple lorsque la probabilité d’événements vasculaires majeurs à 5 ans était inférieure à 5 % la réduction de ce risque pour une baisse de 1 mmol/L de LDL-C était de 38 % (intervalle de confiance [IC] entre 19 et 53 %) contre 21 % (IC entre 16 et 26 %) pour les sujets ayant un risque de plus de 30 % de souffrir d’un événement vasculaire majeur dans les 5 ans. Il faut bien sûr souligner qu’en valeur absolue, le bénéfice à attendre d’une baisse de 1 mmol/L sous statines est bien supérieur pour les niveaux de risque élevés que pour les faibles niveaux avec par exemple 6 événement vasculaires majeurs  évités pour 1 000 sujets à très faible risque (moins de 5 % sur 5 ans) traités pendant 5 ans contre 142 pour 1 000 patients à risque très élevé (supérieur à 30 % sur 5 ans). En d’autres termes, pour éviter un événement vasculaire majeur, il faut traiter 167 patients à risque inférieur à 5 % pendant 5 ans et 67 patients à risque compris entre 5 et 10 %.

Sur le plan de la tolérance des statines, cette nouvelle méta-analyse confirme l’absence de risque carcinologique (risque relatif de cancer de 1 avec un IC entre 0,96 et 1,04), la rareté des myopathies (augmentation de l’incidence de 0,5/1 000 sur 5 ans), le faible accroissement du risque d’AVC hémorragique (+ 0,5/1 000 sur 5 ans) très inférieur à la baisse du risque d’AVC ischémique et évalue le risque supplémentaire de diabète sous statines en prévention primaire à 0,1 % par an (ce qui induirait une augmentation de la fréquence des événements vasculaires majeurs 50 fois plus faible [ + 0,2/1 000 patients traités sur 5 ans] que le bénéfice attendu [- 11/1000] chez des sujets à risque inférieur à 10 % à 5 ans).

**Traiter les hommes au-delà de 50 ans sans bilan complet ?**

A partir de ces données d’efficacité et de tolérance, les auteurs comme l’éditorialiste (2) posent la question de l’extension des indications des traitements par statines. Actuellement pour la plupart des recommandations, il n’y a pas d’indications à un traitement par statines chez les sujets ayant un risque d’événement vasculaire majeur à 5 ans inférieur à 10 %. Or selon l’éditorialiste, le bénéfice à attendre d’une baisse de 1 mmol/L du LDL-C chez ce type de sujets est équivalent à ce que l’on obtient en traitant une hypertension légère, ce que tous les praticiens s’accordent à faire.

Si une telle décision d’abaissement du seuil d’intervention était prise, elle aurait des conséquences importantes en termes de santé publique et d’économie de la santé. En effet, dans un pays développé comme la Grande Bretagne on estime que 83 % des hommes de plus de 50 ans et 56 % des femmes de plus de 60 ans ont un niveau de risque vasculaire supérieur à 10 % à 5 ans.

Au-delà de la question de l’élargissement des indications des statines à la majorité de la population de plus de 50 ans (qui pourrait être économiquement supportable grâce aux génériques), l’éditorialiste va encore plus loin et se pose celle de l’intérêt de se baser sur des bilans complexes et coûteux pour décider d’une prescription de statines qu’il serait peut-être plus pragmatique de fonder uniquement sur l’âge et le sexe ! Un tel algorithme simplissime permettrait selon lui des économies substantielles portant notamment sur l’imagerie vasculaire ce qui serait spécialement utile dans les pays à faibles revenus.

Il reste qu’en pratique, quelles que soient les raisons théoriques d’élargir les indications des statines et l’attitude qu’adopteront les Sociétés savantes dans un avenir proche, de très nombreux patients qui justifieraient une prescription de statines selon les critères actuels n’en bénéficient pas encore (même parmi les sujets à risque élevé) et que l’observance de ces traitements demeure faible (25 % à deux ans en prévention primaire selon une étude par exemple).

Dans l’attente d’un éventuel changement d’indications, les efforts sont donc à porter sur un meilleur suivi des recommandations actuelles et une amélioration de l’observance des patients.

Dr Anastasia Roublev

*1) Cholesterol Treatment Trialists’ (CTT): The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. Lancet 2012, Publication avancée en ligne le 17 mai (doi:10.1016/S0140-6736(12)60367-5).  
2) Ebrahim Shah et coll.: Statins for all by the age of 50 years?  
Lancet 2012, Publication avancée en ligne le 17 mai (doi:10.1016/S0140-6736(12)60694-1)*

**Cestmir Receck:**

Maneuvers provoking reflux in incompetent GSV are based on two different mechanisms:

1.     The compression/release maneuver evokes reflux by reducing the pressure in the lower leg veins, creating thereby a pressure difference between the veins in the thigh and the lower leg. It simulates ambulatory pressure gradient occurring during calf pump activity; principally, it does not influence the diameter of the refluxing channels.

*C.Franceschi: .*

*Even if the proximal pressure is not increased when walking, the reflux is overloaded by the flow/pressure of the calf pump that combined with wall reaction to such a stress leads to dilatation.*

**Cestmir Receck:**

2.     The Valsalva maneuver increases the intra-abdominal pressure and evokes reflux by elevating the pressure in the iliac and common femoral vein above the pressure in the deep lower leg veins; the increase in pressure is accompanied by dilatation of the common femoral vein and the incompetent GSV. This mechanism takes place during coughing, up heaving heavy objects, heavy labor.

*C.Franceschi:*

*OK*

**Cestmir Receck:**

What about to perform both tests simultaneously?

Claude touched on an interesting topic: how to interpret the flow in the tributaries of the GSV arch: is it a normal flow draining venous blood into the femoral vein or is this a refluxing flow escaping from a pelvic vein?

*C.Franceschi:*

*Valsalva maneuver makes the difference.*

**Cestmir Receck:**

In case of a normal flow: the tributary should not be enlarged; no reflux should be detected in the GSV segments both proximal and distal to the issue of the tributary.

*C.Franceschi:*

*in case of GSV incompetence just below the descending tributary while the GSV end is competent, a reflux occurs during the compression-release and Paranà diastole ( more physiological than compression-release , just try it and then let me know.) BUT not during the Valsalva maneuver.*

**Cestmir Receck:**

In case of a flow refluxing selectivelywithin the tributary from a pelvic vein: the tributary should be enlarged

*C.Franceschi: Most of the time, yes , but they are not necessarly very large : thanks to following them up to the “source” of the Valsalva reflux, I “discovered “ the location of some anatomic points so far not described as pelvic escape point ( Perineal (P Point) , Iinguinal (I Point), Clitoridian (C Point) .*

*Cher Cestmir,*

*You ask my “opinion” that I hope to be a “rational answer”.*

**Cestmir Receck:**

1.     The compression/release maneuver evokes reflux by reducing the pressure in the lower leg veins, creating thereby a pressure difference between the veins in the thigh and the lower leg. It simulates ambulatory pressure gradient occurring during calf pump activity; principally, it does not influence the diameter of the refluxing channels.

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**Cestmir Receck:**

; no reflux should be demonstrable in the GSV proximal to the tributary (between the issue and the SFJ); reflux should be detectable in the GSV distal to the tributary.

I would like to ask Claude and other colleagues who have large experience with these provoking maneuvers to specify their opinions on this issue

**Cestmir Receck:**

I did not understand the following sentence… *the reflux is overloaded by the flow/pressure of the calf pump that combined with wall reaction to such a stress leads to dilatation.* Could you please specify in more detail?

**C.Franceschi:**

**The refluxing flow is activated by the calf diastole. Despite the blood is not a perfect liquid, we can apply roughly the Bernouill’s equation, where the pressure gradient is proportional to the hydrostatic gravitational pressure(HGP = mgh) + the static pressure (SP) + dynamic pressure (DP= ½ mv²). So , independently of HGP, SP + DP increases with the velocity and volume of diastolic expansion of the pump. This energy is partly absorbed by mechanical interaction with the venous wall (shear stress and strain due turbulences). This mechanical energy/pressure against the wall leads to a dilatation proportionally to the compliance. This compliance changes with the passive and active structures of the wall. The change of these structures depends more or less on the biologic response to this stress. The overloading flow/pressure is made of the volume/velocity of the blood available in the deep network that refluxes through the SFJ ( or any other incompetent superficial-deep connection). For that reason, the distal disconnection of a closed shunt at the re-entry, ablates the overloading reflux and so reduces the vein caliber. But GHP is not fractionned and continues to exert its pressure against the wall. If the shunt disconnection is proximal, at the escape point, not only the overloading reflux is ablated , but , in addition, the GHP is fragmented, which results in caliber reduction more important than the one that is achieved by the distal disconnection.**

**Cestmir Receck:**

You wrote: *In case of GSV incompetence just below the descending tributary while the GSV end is competent, a reflux occurs during the compression-release and Paranà diastole ( more physiological than compression-release,… BUT not during the Valsalva maneuver.* Can you explain why the reflux is evoked by compression/release maneuver but not by Valsalva maneuver?

**C.Franceschi:**

**In absence of pelvic leak and GSV incompetence, the flow of the descending tributaries cannot reflux neither by the Valsalva manoeuver nor by the compression/release. In case of GSV incompetence distally to a competent terminal valve, the flow of the normal tributaries (included descending tributaries) is evoked because of the inefficient closure of the incompetent valves that allows its aspiration by the pump diastole ( release phase of compression or Paranà).On the contrary, in this case, the Valsalva cannot evoke any descending flow neither in the GSV thanks to the closure of the terminal valve, nor in the descending tributaries because their connections to the deep veins are competent. So only the Valsalva maneuver, when positive, can distinguish a descending reflux fed by a pelvic escape point from a normal descending flow, because the compression-relaxation/Paranà evokes a descending flow in both conditions i.e pelvic escape point/no pelvic escape point. Taking not in account this fact, may responsible of wrong diagnosis and “mysterious” failures.**

**Cestmir Receck:**

I have further questions concerning reflux intensity fed by an arch tributary: Which is the hemodynamic significance of such reflux? How much does it disturb the venous hemodynamics? Which is the clinical relevance of such reflux? Plethysmographic evaluation would clear the situation.

**C.Franceschi: Many varicose veins in female, during and post pregnancy are due to pelvic escape points ( Perineal,Obturator, Inguinal and Clitoridian). The caliber of the refluxing arch tributary is very variable and sometimes decreases to normal and no longer refluxing during the post pregnancy period of time ( reason why I suggest to wait 9 months after pregnancy before treating these varices). When their size is small, the Color Doppler can help fantastically. The clinical relevance is very variable, particularly in terms of trophic disorders, BUT important in terms of cosmetics concern.**

**Cestmir Receck:**

We can suppose that the reflux intensity might be distinctly smaller than in cases with incompetent SFJ; the findings by Cappelli corroborate this assumption: he found out that the diameter of the GSV <5 mm measured 15 cm below the SFJ (which hints at small reflux intensity) was indicative of reflux fed by an arch tributary, whereas diameter larger than 6 mm was mostly found in SFJ incompetence.

**C.Franceschi: Indeed**

**Cestmir Receck:**

As concerns the pressure/flow relations, my approach to discover the motive force triggering reflux was much simpler: I have performed venous pressure measurements and found the peak ambulatory pressure gradient of 37.4 +- 6.4 mm Hg between thigh veins and lower leg veins; Arnoldi found out similar values: 33 +- 11.8 mm Hg. The time behavior of the pressure gradient can be illustrated by overlapping of pressure curves in the popliteal vein and posterior tibial vein (see attached schematic illustration gained from the article by Arnoldi).

**C.Franceschi**

**This is the gradient , i.e DP ( Bernouilly) that confirms my comment. In addition, DUS shows its effect on the flow velocity, direction and regimen**

***In case of GSV incompetence distally to a competent terminal valve, the flow of the normal tributaries (included descending tributaries) is evoked because of the inefficient closure of the incompetent valves that allows its aspiration by the pump diastole ( release phase of compression or Paranà).On the contrary, in this case, the Valsalva cannot evoke any descending flow neither in the GSV thanks to the closure of the terminal valve, nor in the descending tributaries because their connections to the deep veins are competent. So only the Valsalva maneuver, when positive, can distinguish a descending reflux fed by a pelvic escape point from a normal descending flow, because the compression-relaxation/Paranà evokes a descending flow in both conditions i.e pelvic escape point/no pelvic escape point. Taking not in account this fact, may responsible of wrong diagnosis and “mysterious” failures.***

**Cestmir Receck:**

Excuse me, there was a misunderstanding: under the term reflux.

**C.Franceschi**

**That’s why we should agree for the definition of every term in a glossary that takes in account the progress in knowledge. I proposed reflux = flow direction opposite to the valves ….Not every necessary new term should be called jargon.**

**Cestmir Receck:**

, I understand a pathological centrifugal flow leaking out via an escape point from a deep vein (popliteal, femoral, iliac), where the pressure remains unaffected during calf pump activity and where there is enough blood at disposal to counteract the decreased pressure in the lower leg veins. If there is, in case of the arch tributary, no escape point in the iliac vein (Valsalva negative, no connection), we cannot speak about a factual reflux causing hemodynamic disturbance. If a centrifugal flow

**C.Franceschi**

**This is called reflux by the majority of the phlebologists. I proposed retrograde but without success**)

**Cestmir Receck:**

is demonstrable by the compression/release maneuver, the hemodynamic significance of such a flow is negligible.

**C.Franceschi:**

**BUT sufficient to cause varicose dilatations and cosmetic complains.**

**Cestmir Receck:**

I am afraid I must state that the term ***closed shunt*** is a false imagination that does not exist in the reality. The refluxing blood is not a part of a ***closed circulation***as illustrated on your scheme. During muscle relaxation, it is impossible that the blood in the superficial femoral vein moves toward the heart in such an amount to feed the reflux. The blood refluxing in incompetent GSV comes from ***incompetent***iliac and common femoral vein where there is enough blood at disposal to counteract the decreased pressure in the deep lower leg veins. You can try to prove it using color coded DUS. There must be enough blood at disposal to evoke hemodynamically efficient reflux; this venous blood stock can be supplied only from incompetent deep veins; any competent valve situated above the escape point in the deep vein reduces essentially the supply and makes the resulting reflux hemodynamically hardly efficient.

**C.Franceschi**

*During the pump diastole, the pressure gradient between Sperficial-femoral vein and calf re-entry is favorable for a superficial femoral vein emptying trhough  the incompetent GSV ( closed shunt). The GSV reflux is also fed by the iliac vein ...but only when the iliac and common femoral valves are incompetent (first case).When they are competent (second case) , the reflux is also important even if less important than in the first case. I saw it with Color DUS.*

**C.Franceschi:**

Open shunts and open deviated shunts presented on the schemes do not evoke a pathological reflux that would be able to cause hemodynamic disturbance: they are hemodynamically ineffective because the amount of the supplied blood is not large enough to cause hemodynamic disturbance: it comes from the capillaries.

**C.Franceschi** *Open Vicarious shunts are not involved in diastole but only permanently ( reisdual pressure from the capillaries)  with peaks evoked by the muscular systole. The Open deviated shunts are superficial incompetent tributaries that are overloaded during the diastole by the GSV flow ( made of the flow coming from its competent tributaries). Indeed, this shunt is not overloaded as much as when coming from the deep veins, but it exists though its hemodynamic relevance is not sufficient to create important pressure disturbance but it is suficient to dilate the superficial vein ( varicose) .*

**Cestmir Receck:**

Thus, you have a turn to prove (using plethysmography or venous pressure measurements) that open shunts and deviated open shunts are hemodynamically effective; until then they must be considered hemodynamically inefficient.

**C.Franceschi:**

*I didn't perform such measurements BUT the vein can be considerd as a gauge because its caliber change ( clinically and DUS) with the pressure.*

**Cestmir Receck:**

Air plethysmography measures functional venous volume: this is the volume change that occurs from the supine to the standing position. It is fed physiologically by venous flow coming through capillaries;

**C.Franceschi:**

*Yes, the residual pressure ( term usually used in physiology)*

**Cestmir Receck:**

in pathological situations also by reflux

**C.Franceschi:**

*Yes*.

**Cestmir Receck:**

Open shunts  and deviated open shunts represent the filling that takes place physiologically through capillaries

**C.Franceschi:**

*Yes BUT the physiological capillary flow is drained by the wrong vein where into which it is diverted, permanently with  systolic peaks through the open vicarious shunts and during the diastole through the open deviated shunts.*

*Thank you again for your interest in my studies, because your expertise helps me to improve my explanations.*

*Attached a model and its physical rational validated by the Physics Lab of the French Ecole Polytechnique and a physicist of the Polytechnique de Lausanne..*

**Cestmir Receck:**

There is indeed a pressure gradient between the superficial femoral vein and the deep lower leg veins during calf pump activity but there is no incompetent communication between these pressure poles (if we exclude e.g. mid thigh perforator) to trigger reflux. The GSV issues into the ***common femoral vein***. Reflux (diastolic centrifugal flow) within the incompetent GSV is **exclusively**fedfrom from the incompetent iliac and common femoral vein.

**C.Franceschi:**

**BUT there is a path through the incompetent SFJ that make it possible (ask any physicist) I saw ( Color DUS) this also in a deep closed shunt made of a double Superficial femora vein. You are right , in case of iliac-common femoral vein incompetence ( Trendenburg described it ) the reflux is eased by the gravitational pressure and is predominant to the superficial femoral vein supply. Another evidence is that in case of iliac thrombosis ( while the popliteal vein is competent) associated to GSV incompetence , an important reflux ( I have seen it several times) . On an other hand, a closed circuit ( closed shunt, private circulation) can be also defined as a circuit that allows part of deep blood propelled up by the systole through a vein (e.g up to the iliac) , to be aspirated back during the diastole through another one ( e.g GSV) , and so on repeatedly at each step when walking.**

**Cestmir Receck:**

Thus closed shunt/closed circuit to all intents and purposes does not exist.

**C.Franceschi:**

**Thus closed shunt/closed circuit to all intents and purposes does exist.**

**Cestmir Receck:**

If there is a competent valve in the CFV or iliac vein (a very rare situation), only the venous volume contained between the competent valve and the SFJ is available to counterpoise the decreased pressure in the deep lower leg veins; this volume is to small to cause a significant hemodynamic disturbance.

**C.Franceschi:**

**Unless it is bigger because fed by the superficial femoral vein ( closed shunt)**

*The Open deviated shunts are superficial incompetent tributaries that are overloaded during the diastole by the GSV flow ( made of the flow coming from its competent tributaries).*

**Cestmir Receck:**

Where is the source of reflux in open deviated shunts?

**C.Franceschi:**

**At the junction between the GS trunk and the incompetent tributary**.

**Cestmir Receck:**

You often speak about ***overloading;*** how do you measure overloading? In which units?

**C.Franceschi:**

**Velocity and time of the reflux at the escape point as displayed by Doppler .**

**Cestmir Receck:**

Which is the value discriminating overloading from not overloading?

**C.Franceschi:**

**Presence or absence of escape point e.g a refluxing tributary disconnected from the escape oint is still refluxing but its flow is reduced to the draining flow fed by its skin territory.**

With CHIVA, what is the relative success rate of ulcer cure in the patient with both deep and superficial reflux venous reflux compared to the patient with superficial reflux only?  
  
Tom Eaton

Dear Tom,

It depends on the severity of the deep reflux ( for which I proposed a rating Total, partial and segmental in our book Principles of venous hemodynamic Franceschi,Zamboni Novapublishers NewYork 2010).).

When the reflux is total, the maneuvers cannot evoke any reflux in the GVS even if even if varicose wiith destroyed valves because the pump diastole aspiration is inefficient ( the pump doesn’t play its role if the muscles doesn’t work and or the deep valves are destroyed ( like the heart ). Because the deep reflux in that case “competitive”. A century ago, Perthes invented a test that predicted the inefficacy of the GSV ligation when placing a tourniquet at the thigh, the GSV didn’t collapse below and that relates to this phenomenon of competitive reflux. So the more the deep reflux, the less the superficial reflux. Testing the deep veins is not sufficient and must be completed by the Perthes test. So, the result of GSV ligation will depends on the grade of the deep reflux. If the grade is partial or segmental, a GSV reflux is seen ( even if less important) and the result will be more or less satisfactory.

In our practice, we treat before the deep reflux when it is possible ( in case of deep venous closed shunt). The reappearance of an important GSV reflux is somehow a good “evidence” of the efficacy of the deep CHIVA. In that case , the superficial CHIVA of the GSV is performed, as a second step of the treatment. If deep CHIVA is not possible ( no deep closed shunt) and the deep reflux is totally competitive ( no diastolic GSV reflux), we treat it medically.

I hope I gave you a satisfactory information.

Claude.

**Dear Raj,**

**Here is my response in blue,**

I picked up this idea of yours on excess TMP from your book and I found it intriguing particularly with regard to a possible mechanism to explain the efficacy of compression. Mayrovitz and Macdonald in an article in International angilogy showed that arterial perfusion increases in response to possibly reduced TMP with compression. **n that study, the pulsatility of the flow was assessed by nuclear magnetic resonance flowmetry and has shown that forefoot-to-knee compression bandaging (40 mmHg) caused a highly significant (P<0.001) increase in the bandaged leg pulsatile blood flow due to increases in both peak flow and pulse width . The compression by a cuff , as done decades ago, was use for arterial Oscillometry assessment, where the oscillation ( peaks ) increases with the cuff pressure. That could be an “artifact” of explanation, because the pulsatility could be due to the arterial compression instead of the venous. . On the other hand, the effect of the compression on TMP is double. It decreases the TMP of the skin microcirculation in proportion to the strength of the compression ( Starling), which accounts for edema reduction and ulcer healing. …. On the other hand we can stipulate the hypothesis that the paradox of a mix of red blood and necrosis could be due to the capillary arterial flow stop ( excessive intra-venous pressure) while the AV micro-shunts opens ( reflex? Inflammation?) reducing the micro-circulation resistance, thus increasing the Residual venous pressure, and responsible for the “red” bleeding ( a study that I don’t remember the authors, has detected a high HbO² at the level of the venous ulcers).**In an experimental model (see JVS-VLD web under "in press"  an article on 'Critical venous stenosis' **I didn’t find it, could you mali it?)** increasing the extravenous pressure did not decrease TMP; intravenous pressure simply rose to equal the external pressure applied with the result there was no change in TMP. The increase was apparently due to conversion of velocity to pressure energy per Bernouli. **I have not enough information about this study and I cannot answer you precisely. Nevertheless, the pressure in the superficial veins is necessarily higher than in the deep, otherwise, the superficial blood wouldn’t drain, which explains why an external compression reduces more the caliber of the deep veins than the superficial..** However, there is not much velocity in the superficial veins near the ankle for such conversion to take place **Obvious Raj**. Perhaps TMP does fall unlike in the experimental model. It will be crucial to know whether this has ever been measured in patients?

**Amitiés**

**Claude**

**Dear Raj**

You write: Increasing the extravenous pressure did not decrease TMP. intravenous pressure simply rose to equal the external pressure applied with the result there was no change in TMP. The increase was apparently due to conversion of velocity to pressure energy per Bernouli

**Claude: In this experimentation, the Pressurizing reservoir (11) measures ONLY variations of the pressure/volume of the Penrose and NOT external pressure. The external pressure is given by the volume/weight of the Pressurizing reservoir, that doesn’t vary in this experimentation. So, the measure of the TMP by the difference between Penrose pressure and the pressure shown by Pressurizing reservoir, is wrong and will not change with the changes of the Penrose pressure. . In fact, the TMP is the difference between the Penrose pressure and the pressure provided by the volume/weight of Pressurizing reservoir. When the downstream resistance is low , the TMP decreases. It is the case when the compression at the leg is applied in normal or incompetent veins in absence of downstream high resistance. In case of downstream high resistance, before compression, the TMP is high because the IV pressure is high due to accumulation of Residual Pressure. Compression will increase the external pressure and will decrease the TMP in reverse proportion to the downstream resistance (that increases the lateral pressure).**

**This is a typical case of mistakes that occur when the “physical initial data” are not clearly defined.**

**I hope I have been understandable despite my broken English.**

**Amitiés.**

1. Mechanical venous model. (A) Shows the Penrose

enclosed within a Plexiglas cylinder. Various parts are shown in the

schematic (B), 1. Inflow reservoir, 2. Inflow tubing, 3. Upstream

pressure monitoring catheter, 4. Penrose pressure-monitoring

catheter, 5. Penrose tubing, 6. Plexiglas cylinder, 7. Short

connector in between Penrose and tubing, 8. Ball valve used to

simulate outflow stenosis, 9. Outflow tubing; level of outflow

orifice could be varied; 0 mm Hg and 10 mm Hg are shown;

outflow tubing size varied from 1/2 inch to 1/8 inch (shown at

the bottom) to simulate outflow stenosis, 10. Outflow tank, 11.

Pressurizing reservoir for the Starling resistor.

Many regards,

Raj

I should correct myself and say that resting pressure (gsv & deep) will be the same but injected contrast flows out preferentially through GSV due to lower resistance rather than through slightly higher resitance perforators (related to size) into the deep.

Raj

**A MRI study ( article Veins and Lymphatics Rhab and all) as shown that at rest, the compression shrinks more the deep veins than the superficial , which advocates for a GVS pressure > Deep veins pressure. Anyway, the pressure at the GSV end is necessary ( physically mandatory) to the GSV drainage into the femoral vein. The difference may be small, but sufficient to create an efficient pressure gradient.**

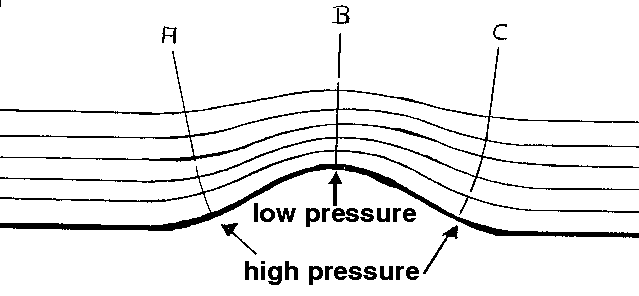
L’équation de Bernoulli simplifiée spécifie que le gradient de pression maximal (∆P) est égal à 4 fois la différence entre le carré de la vélocité maximale dans la sténose (V2) et le carré de la vélocité maximale d’amont (V1, chambre de chasse) :

∆P = 4 · (V2VAo - V2CCVG)

OK, but Bernouilli equation is not a yesterday discovery.

P(a) + 1/2 rho v(a)^2 + rho g h(a) = P(b) + 1/2 rho v(b)^2 + rho g h(b)

In this picture you can see the consequence of the fact that energy pressure  (**P** in the above-stated equation) is only part of the total energy of moving fluid.



Marian Simka

**Dear Marian,**

**Your picture and calculation ( see below) is not appropriate to the Pressure gradient PG in the veins. You show the intrados/extrados of a plane wing moving in steady air (fluid) , so you cannot have a pressure gradient between A (back edge of the wing) and C (front edge of the wing) and Total Pa = Total Pb is null, thus PG is null. So the initial conditions are different from fluids in a conduit, i.e . in the veins where the initial conditions are opposite, because the fluid moves and the veins are steady. The Pressure Gradient is a derivative function that takes in account the difference of the total pressure of the fluid and the distance between 2 spots of a conduit.**

**If the blood moves, there is necessarily a pressure gradient PG between 2 spots of the vein a and b, and the PG depends on dP = Total P a and Total P b. As you know, Bernouilli equation is:**

**Where P = lateral static energy/pressure , ½ mv² = kinetic energy/ pressure ) and gh = gravitational energy/pressure**

**PG depends on dP that is = Total Pressure a – Total Pressure b = ( Pa + ½ mv²a + gha ) – (NOT =) ( Pb + ½ mv²b + ghb )**

**SO, as (P + ½ mv²) is constant, any variation of P is compensated by the opposite variation of ½ mv². Thus, your reasoning do not comply with the fluid mechanics laws, because of a misinterpreted Bernouilli’s equation.**

**BTW, our communication needs not only an effort for improving English ( I try but still with bad results) but also to improve our knowledge in fluid mechanics ( I try to understand some with help of the physicist of Polytechnic, since decades…)**

**BW**

Dear Claude,

All your statements are correct and I cannot find fault with them.

I hope the following support your understanding.

**Dear Chris,**

**Allow me to add some “in blue” at your statement in** “black”

1) Pressure gradient reversal is mainly a function of activity in disease - NOT at rest in normal legs.

**In disease by incompetence, No difference with normal legs at rest , lying or standing still ( no muscular pump activation) except a slight difference due to the effects of the thoraco-abdominal pump. The difference arises WHEN walking, where the valve incompetence cannot provide a proper dynamic fractioning of the gravitational pressure DFGP and activates closed and deviated shunts because, incompetent, they cannot block the reverse flow evoked by the muscular pump diastole .**

2) In health there is very little blood in the saphenous veins (it takes too long for them to fill) so there is not a lot to enter the deep system.

**But the few that enters is crucial for the skin drainage. If you bock this inflow, the superficial veins dilate due to the Residual pressure increasing the TMP and it dilates the caliber , opens collaterals in order to by-pas this obstacle (e.g recurrent varicose post stripping/phlebectomy) and impairs the drainage ( edema, trophic disorders)**

3) At rest in refluxive saphenous trunks arterial drive-though takes over. Hence the presence of a saphenous pulse

.

Lattimer CR, [et al.](http://www.imperial.ac.uk/AP/faces/pages/read/Home.jsp?person=c.lattimer09&_adf.ctrl-state=d5vehk7p0_3), 2012, Saphenous pulsation on duplex may be a marker of severe chronic superficial venous insufficiency, Journal of Vascular Surgery, Vol:56(5), Pages:1338-1343

**The excess of TMP due to Saphenous overloaded reflux provides distal inflammation that reduces the Microcirculation resistances ,so that the pulsatile arterial inflow is less amortized in the microvessels ant the venous outflow pulses in reverse proportion to the microcirculation resistances**.

4) Superficial venous insufficiency CAUSES an in-balance between the deep and superficial systems so the pressure in the latter is greater.

…**.Yes but only during the muscular pump diastole, when the deep outflow into superficial triggered by the PG reversal prevented by valve closure in normal, is not prevented in disease. For that reason, a venous valve incompetent individual is NORMAL if he stops moving his muscles ( varicoses veins collapse then remain so after paralysis, like hemiplegia or paraplegia).**

I don't think we have any differences of opinion.

**I have just pointed out some crucial issues.**

**Tres amicalement**

**Claude**

 Claude,

Thanks for such extraordinary efforts for the colleagues who are interested to learn but afraid(?) to ask.

Such self-explanatory animation diagram should give a substantial clearance to what the CHIVA actually means to the clinicians/phlebologists.

***Dear BB and all,***

**BBLee**: I would like to initiate the questions to stimulate/encourage others. How long do you wait/interval between Step 1 and Step 2 for Shunt II?

***I think you mean SHUNT III and not shunt II (number mismatch). 2 optional strategies in Closed Shunt type III: ( Example when regards the GSV): SFJ diastolic reflux ( N1>N2 escape point) from the femoral vein (N1) descends along a more or less extended incompetent GSV (N2) , crosses a second escape point N2>N3 then proceeds along an incompetent tributary (N3) down to a re-entry perforator (N3-N1), so back to the deep network (N1). No intermediate visible or effective re-entry on the incompetent track of the GSV ( The presence of such an intermediate re-entry produces another pattern, i.e the combined Shunt I + II)***

***Option N°1: - CHIVA 2 steps consists of***

***1st Step : incompetent tributary-GSV junction (N3-N2) flush ligation/disconnection that results in:***

***-Gravitational pressure fraction at the N2-N3 escape point level but leaves behind above a high Gravitational pressure column***

***- GSV reflux ablation ( centripetal flow restore) and***

***-N3 reflux no more overloaded by N1 and N2 flows, drains the N3 territory into N1, so physiologic in terms of function ( correct drainage hierarchy) despite non physiologic but non pathologic in terms of direction..***

***2d Step: SFJ flush ligation/disconnection, when , after a 6-12 months period of time, the high gravitational pressure left behind above the N3-N2 disconnected junction has opened a perforator on the GSV trunk producing a SHUNT 1 ( which occurs in almost 85% of the cases (see Zamboni’s study)..***

***Option N°2: CHIVA 1 step only: SFJ (N2-N1) ligation/disconnection + incompetent tributary-GSV junction (N3-N2) ligation/disconnection + Devalvulation of the GSV competent valves below the N3-N2 junction down to a “good” perforator in order to achieve a N2>N1 re-entry.***

**BBLee** :When do you do the simultaneous ligation- devalvulation- for Step 2 among Shunt II and accept the thrombosis and its indication?

***Claude: Simultaneous ligation N2-N1 and N3-N2 is conservative BUT not hemodynamic because the previously GSV reflux is BLOCKED and no re-entry being available, the GSV thromboses and lead to recurrences by OVS effect. This thrombosis acceptable because not dangerous because disconnected from the deep network, but it represents a failure in terms of hemodynamic/functional treatment. Devalvulation down to a –re-entry perforator prevents it because it produces a draining re-entry. Some times a clot occurs, but disappears quickly ( some weeks) and leaves behind a flowing and structurally correct GSV ( JM Escribano CHIVA meeting 2012).***

***By the way, the CHIVA strategy in SHUNT II (open deviated shunt) consists of a one step only procedure , as ligation/disconnection of the refluxing tributary flush its connection to the GSV (N3-N2).The rate of recurrence is low around 5% ( still Zamboni’s study).This SHUNT II represents also the only configuration where ASVAL is efficient.***

***I hope I answered you questions and I have given some additional information for a better understanding in SHUNTS as animated in my pps.***

***I feel like a fellow of a small group of men lost in a desert island who throws several bottle-letters in the sea since decades and sees a rescue ship sailing towards us , skipped by Captain BBlee.***

***Amitiés***

***Claude.***

Hope this could bring more questions and further more interest/understanding on CHIVA.

Congratulations!

BB

Chris

Dear BB,

When there is a pressure gradient (P1&P2), there is flow and the velocity increases when P2 is lowered. That is because, Information about P2 is constantly transmitted back to P1 by pressure waves and these have a characteristic "wave speed" . Wave speed can vary and can be slow in veins when there is vein collapse. P2 information does not get back to P1 when flow velocity is extremely fast ('critical velocity'), the wave speed is slow or both. In vein collapse,  the flow velocity increases because of the narrow flow channel and the wave speed decreases as well. In that case, flow velocity will continue at critical speed  and any change in P2 has no influence. Engineers call it choking. A  waterfall is another example, lowering the river level below will have no influence on waterfall discharge. Such a phenomenon was shown by the great Carl Fung (considered the father of bioengineering in US) in lung flow and has been posthulated in the kidney and in precapillary circulation. Such a phenomenon has been proposed in IVC at the diaphragm level. Veins and other collapsible tubes  are known to collapse just beyond compression points such as fascial entrance, after sphincters (urinary stream) and IVC passing out of pressurized abdomen into thorax. Many of these are still controversial except perhaps lung flow.

**This back pressure wave speed pattern is the reason for the increase of the central arterial pressure in proportion to the arterial stiffness, the too fast back ( centripetal) wave adding its pressure to the following systolic centrifugal wave at the output of the heart. Applied to the collapsible tubes and metaphorically waterfall , this pattern for the critical speed effect in collapsed veins is interesting, but not that much convincing. Therefore, another hypothesis can be proposed as the critical velocity closure of collapsible tubes as veins. The speed is high thanks to low downstream resistance. According to Bernouilli, in case of high speed the total pressure can be totally converted into dynamic energy so that the lateral pressure is null , so producing a negative transmural pressure ( because the external pressure is not null) and the tube collapses and closes This closure cannot last because the closure stops the velocity and the earlier effect stops at the same time. So the upstream pressure reopens the tube, the velocity increases and once more time, the vein collapses once again and so on .. This phenomenon produces vibration of the tube and can explain e.g snoring …and why not vocal cords vibrations ( voice…). On the other hand, the micro-vessels phasique flow detected by Laser Doppler could be related to another phenomenon I won’t discuss here.**

**Dear Tom and all,**

**Tom, I appreciate your positive skepticism about CHIVA outcomes. As promoter of this method, I was myself so distrustful in my objectivity that I let other international groups perform long term follow up RCTs. these RCTs validated the "paradoxically" CHIVA prevail on the gold standards stripping and compression (1,2,3,4), the confirmed by a Cohrane review (5). You may not be confident in these evidences...but were Annals of Surgery, JVES and Cochrane Librairy mystified by so many fake/dishonest studies?**

**In addition, a Chinese study reports CHIVA better than Laser (6) and another study (7) shows that CHIVA is better than stripping when performed by who they call “CHIVA experts” and worse when performed by who they call “CHIVA non experts” pointing out a necessary minimal knowledge about the method.**

**Best wishes**

**Claude**

**[1] Carandina S, Mari C, De Palma M, Marcellino MG,Cisno C, Legnaro A, et al.Varicose Vein Stripping vsHaemodynamic Correction (CHIVA): a long term randomised trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230–7**

**[2] Parés JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX,et l.Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624–31**

**[3] Iborra-Ortega E, Barjau-Urrea E, Vila-Coll R, Ballon-Carazas H, Cairols-Castellote MA. Comparative study oftwo surgical techniques in the treatment of varicose veins of the lower extremities: results after five years of followup. Estudio comparativo de dos técnicas quirúrgicas en el tratamiento de las varices de las extremidades inferiores: resultados tras cinco años de seguimiento]. Angiología 2006; 58(6):459–68.**

**[4]P.Zamboni and all: Minimally Invasive Surgical management of primary venous Ulcer vs. Compression Eur J vasc Endovasc Surg 00,1 6 (2003)**

**[5] Bellmunt-Montoya S, Escribano JM, Dilme J, Martinez-Zapata MJ. CHIVA method for the treatment of chronic venous insufficiency. Cochrane Database of Systematic Reviews 2012 , Issue 2 . Art. No.: CD009648. DOI:10.1002/14651858.CD009648 .**

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**[7] Milone M, Salvatore G, Maietta P, Sosa Fernandez LM, Milone Recurrent varicose veins of the lower limbs after surgery. Role of surgical technique (stripping vs. CHIVA) and surgeon's experience.F. G Chir. 2011 Nov-Dec;32(11-12):460-3**

Dear Claude, ( ppt compression)

Fantastic! Spent quite an amount of time to read through this afternoon and enjoyed enormously.

To compliment once again for your enormous efforts, I wish to make a small comment/contribution you might consider to accommodate when you make a next revision as following;

1.  I humbly suggest you to use the term/vocabulary 'precipitate' to replace current term 'cause' if you would. I am sure no one would object to current description with 'cause' since it is true but to consider all other factors, 'precipitate' would give a bit more room to evade the frontal attack/criticism.

2.  I also suggest you to add 'gradient' to 'transmural pressure' to make it as 'transmural pressure gradient', which seems to be more helpful for clear understanding according to my (biomed engineer) graduate students. Without the word 'gradient', it is sufficient to deliver your intention but certainly this change would give more weight.

Dear BB,

About your kind comments about my ppt Compression. Here are my comments about yours.

1- OK for precipitate…

2- Transmural Pressure is physically and physiologically different from Pressure Gradient.

A-**Transmural pressure** **TMP**:

1- **Physically** , TMP is  defined as the pressure inside the container minus

the pressure outside the container, e.g the vessel walls. **Only part of the intra-venous pressure is involved in the TMP**, i.e the gravitational and static/lateral pressure (Bernouilli..once again WHO have stolen “his” theorem from his son) .

2- **Physiologically**, TMP is crucial for the **drainage of the tissues and the cardiac function.** Indeed, in veins TMP determines the caliber/volume of the venous bed ( involved in the reservoir effect as regulator ( capacitor) of the Right ventricle pre-load). Beside this effect, TMP is crucial in the venous capillaries bed where it determines the drainage (with the other factors as osmotics as defined by Starling) .

**B- The Pressure Gradient (PG) is** Physically and physiologically different from TMP.

**1-Physically:** A Gradient is a vector sum of the partial derivatives with respect to the three coordinate variables x, y, and z of a scalar quantity whose value varies from point to point, i.e change in the value of a quantity (as temperature, pressure, or concentration) with change in a given variable and especially **per unit distance in a specified direction.** PG in vessels, is the **vector of scalar value of pressure of a fluid in continuity between 2 points** ( not between both sides of a fluid discontinuity as a wall). **The inside venous pressure involved is not a part ( as TMP) but the whole of the pressure.**

**2-** **Physiologically:** PG doesn’t interfere directly with the venous bed volume and drainage , but it is the **determinant factor of the venous volume flow.**

**These definitions are not MINE but those anybody can find in various dictionaries. SO, I think that it is more rigorous to keep both terms.** (I teach also biomed engineers, beside angiology to MD.)

**3- Pressure is negative when lower than the atmospheric. It is the reason why introducing a needle in a jugular vein in seated patient is dangerous because the venous pressure is lower than the atmospheric and air enters, responsible for air embols. In upside down position, the legs are in the same conditions as the neck in seated position..**

**4- I ignored intentionally multi-chambers because it doesn’t change the rational of compression.**

**6- Compression in AVF is efficient according to its location. E.g a compression of finger affected by AVF, is the best “accommodation” in my experience: Stop of the diastolic flow due to the AVF and progressive reduction of the AVF with time (months). On the contrary, a young girl (12 years) affected of a high flow of multiple thigh AVF, compression won’t change because inefficient. BUT in that case, the girl had previously successive embolizations with immediate cutaneous necrosis (bleeding) spot and fast recurrence. I gave her compression for the bleeding cutaneous ulceration.**

**7- No suggestion about starling low for capillaries .**

**Thank you BB for “forcing” me to write/improve my English as well as my "communication"  which sticks me to my study, so preventing me to sin i.e enjoing the fine weather in my country house ( Burgundy France) and walking in the surrounding forest where delicious  mushrooms are waiting to be collected.**

**Amitiés ( which means “friendship”)**

**Claude  .**

3.  In my opinion, the negative (-) intraluminal pressure on upside down position is somewhat distorted - perhaps you did it intentionally to emphasize on the gravitational pressure-. It should be basically same as the pressure gradient along the arm elevation above the heart level/phlebostatic axis so that it shouldn't go to the minus level but stay on the positive (+5mmHg?). Opinion?

4.  I wonder whether you ignored intentionally on current pneumatic pump structure with multiple chambers to provide sequential increase of the pressure to more dependent part, so called 'Compartmentization', saying ' ---- compression pressure not uniformly distributed---"?

6.  I have a strong reservation on your statement on the role of compression therapy to 'AV fistula'. There is NO room to accommodate the bandage and all the modalities of compression for the management of AV fistula either congenital or acquired; it only gives a false sense of security deferring  mandated  care of the AV fistula itself. The attempt to control of venous hypertension would make the condition of AV fistula- BTW, AV shunting 'fistula' is a bad/wrong term for the truncular form of the AVM with no nidus (e.g. pulmonary AV fistula) ISSVA Classification erroneously uses, because ALL the AVM lesions are 'fistulous'!!!!- worse.

7.  I have been giving the class to the graduate students starting with the introduction of Starling's Law for decades. But more I look into this creed(?) from the 'microcirculatory homeostasis' point of view, more uneasy I feel since we are missing/ignoring one critical role of the lymphatic drainage as an active participant to this homeostasis, and not as a bystander. Any comment on this potential liability of classic Starling's Law since you vaguely touched/embraced the lymphatic issue, on and off, throughout the presentation?

Thanks for the PPT you kindly shared with us.

With respect,

BB

**Physics, fluid mechanics, hemodynamics: terms and definitions**

**Hemodynamics obeys the fluid mechanics law which is part of physics. Thus , hemodynamics terms and definitions must relate to physics laws, terms and definitions. Terms can be whatever, eponyms e.g Ohm’s law, Ampere, Special Relativity ( called Invariance in 1905 by Einstein because related to the light speed invariability then changed in Special Relativity by Max Planck in reference to Galileo’s concepts ) which are just “labels” consensually accepted by the scientific community to design scientific principles, concepts and laws.**

## 1-Energy: Energy is the capacity (potential or active) of a physical system to perform work, i.e the integral of the force over a distance of displacement (Newton x meter or Joule) and can be quantified just measuring the performed work. Energy can transform into several forms such as potential and kinetic energy, pressure, heat, electricity, light, and so on.

## 2- Fluid Pressure ( Total, Potential, kinetic, lateral) is a peculiar form of energy that is the result of a force exerted per unity area.

## 3- Gradient: A Gradient is a vector sum of the partial derivatives with respect to the three coordinate variables *x, y,* and *z* of a scalar quantity whose value varies from point to point, i.e change in the value of a quantity (as temperature, pressure, or concentration) with change in a given variable and especially per unit distance in a specified direction.

**4- Pressure Gradient is a peculiar form of Energy Gradient. Pressure Gradient in fluids is the vector of scalar value of pressure of a fluid in continuity between 2 points. So Pressure Gradient is a member of Energy Gradient family.**

**Beliefs, intuition, rationality, science and varicose veins**

**Beliefs and intuitions are necessary to science as hypothesis, but they do not become scientific as long as they are not verified with the scientific experience/trials. When you do not believe in facts scientifically validated by studies, RCTs, Cochrane reviews (ref below: 1,2,3,4,5,6,7) , we must either provide evidences that contradict them Scientifically, or demonstrate that the evidences are false ( fraud, errors, deceit). Some evidences may also be made by visiting places where the suspected methods are practiced or even watching the videos that show the “unbelievable” varicose veins collapse though not destroyed.** [**https://dl.dropboxusercontent.com/u/40615437/CHIVA%20CACVS.wmv**](https://dl.dropboxusercontent.com/u/40615437/CHIVA%20CACVS.wmv)

<http://www.youtube.com/results?search_query=chivachannel>

**Suspicion is mandatory in science because any theory or method is not the final truth/reality, just the current best representation of the truth/reality that has to be improved/overcome by other theory or method. So suspicion about CHIVA is productive if, confirmed by scientific evidences, it leads to a better one in terms of coherence and efficiency.**

**So, Tom and Paul’s unbelief will improve the scientific and efficient knowledge in Phlebology when they transform their unbelief in scientific evidences.**

**Claude**

**[1] Carandina S, Mari C, De Palma M, Marcellino MG,Cisno C, Legnaro A, et al.Varicose Vein Stripping vsHaemodynamic Correction (CHIVA): a long term randomised trial. European Journal of Vascular and Endovascular Surgery 2008;35(2):230–7**

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Asval is easy to understand by us , obtuse vascular surgeons. Yes we did it before it was conceptualized but Rea's and Paul's studies gave us comfidence when proposing this treatment to our patients.  
> Chiva may be an option but few of us can master the ultrasound examination to the high level required, so we prefer no Chiva to a bad Chiva.  
> This is real life, and many more surgeons use Asval than Chiva.  
> It may change with younger generations- time will tell.   
> Meanwhile, thank you Paul not only for Asval but for your open mind and organizing skills.  
> Regards to all.

Cher René,

We know each other since 1975 when I struggled for my findings in Doppler US Semiology against the majority because too difficult and not reliable, and definitely accepted decades later…

Nobody is against Paul. The issue is just a discussion about scientific rigor in terms of venous disease. You point out judiciously

NOBODY’S PERFECT : Rhetoric , sense , and ethics

Dear colleagues,

I’ll try to comment rationally the mails related to the opinions about the efficiency of the various venous treatments.

1- Telling just “ No method is perfect” is a rhetorical trick to make believe that all methods are equally imperfect, i.e “ to drown the fish (French idiom)”. Sensible/logic statement should be “No method is perfect, but some are less imperfect than the others” and cite them not only according to the personal conviction but also according to the scientific data. .2- Telling just “No surgeon is perfect” relates to the same issue.

2- Telling just: “We perform not the current best validated method if we don’t know/understand it properly” is against the Hypocrate’s oath because we are supposed to offer the patient the best current available treatment.

3- Telling just: “ We may not perform the best validated method because it is difficult to understand/perform”. Is this a valid excuse? This is contrary to what specialists/experts/professionals are supposed to be, i.e professional who make the sufficient efforts even if tough to acquire the sufficient knowledge. Who, among the anti-CHIVA made this effort? Never saw them attending my courses and workshops ( except ACP ).I’m not invited in the French meetings and congresses in France ( why isn’t it Paul? Why isn’t it René?) and the “back stage” ethically/professionally disputable information to students and others is “CHIVA is obsolete and recurs promptly” in contradiction with the international studies they “forget” to quote. BTW, in Spain, where it is taught at the university to Vascular Surgeons, CHIVA was performed in around 40% of the patients ( Public register). Remember René we met for the first time on 1976, when I published the first world book about CW Doppler semiology and technics resulting from my studies ( Investigation vasculaire par ultrasonography Doppler Masson edit translated in Italian and Spanish) , how many opponents stated it “not reliable/ too difficult” and 2 long decades later accepted as evidences.

4- The final sentence “ Nobody’s perfect” of the movie “Some like it hot” is a fantastic and humoristic example of rhetorical way to hide an uncomfortable fact.

Regards

Claude

Dear Tom, and colleagues who are concerned by the the saphenous Preservation/destruction

Pertinent question, Tom. Here are 2 statistics made by non-CHIVA fans nor defenders:

1- Probability of a necessary arterial by-pass after venous surgery : 3% (1)

Venous surgery : 200 000 / year **+** 6000 000 sclerotherapies: .

2- Venous aorto-coronary by-passes: 70% among the varicose presented venous segments eligible for ACBP (2)

I’m sure you trust in the authors of these studies.

(1)  Lofgren EP. In Bergan JJ, Yao JST (eds). Surgery of the veins 1985 285-299

(2) Cohn et al, Ann Thor Surg 2006 81(4) 1269-4

My personnal statistics: I studied the possibility of GSV preservation because "too often"we had concerns and catastrophies  about patients who needed a venous graft for arterial  by-pass while their GSVs missed because previously drastically "treated" for varicose veins.

For my ethics as a humùan being multiplied  by my MD responsability, such a risk, is   sufficient, whatever the probability,   to treat properly "billions of patients " (Isn't it  Alessandro ).  ,

No, Alessandro, my baby  isn't CHIVA but any singular patient of the "billions" you quoted  . I gave the acronym CHIVA instead of Franceschi's method in order to patent it no and avoid another eponym ( isnt' it Malay?)

Amitiés

Claude

The presence of a draining saphenous axis, whatever its flow  direction :

1. Allows a good drainage of superficial tissues, so preventing  the appearance of telangiectasias or blue reticular veins refractory to sclerotherapy
2. Confines  the reflux fed by  new or left behind escape points as  SFJ , Hunter perforator, pelvic escape points, in an approriate vein  , instead of in anarchic neo-varicose veins .
3. Lets of  re-do surgery when  the shunt overload is innocent in terms of symptoms and signs as drainage impairment and visible varicose vein as shown in the reprted case. .
4. While an easy   re-do mini-surgery if feasible if this recurrence is responsible for recurrent symptoms and or signs.

 In Conclusion the preservation of the  saphenous trunk  eases the management of the recurrence or evolution of this chronic disease and make our patients satified

Tom,

In France, the first choice for coronary artery by\_pass is not only the left but also the right mammary ( we assess their caliber at their origin by DUS and still by DUS we check up their post operation patency: typical camel l shaped velocity curve). Then the radial artery. BUT the saphenous vein is used in case of complementary/multiple coronary by-passes (recently my brother in law after stent failure, and an anesthetist colleague who suffered a CHIVA procedure 10 years ago)..

In France, peripheral venous by-passes , particularly below the knee are mandatory when the endovenous procedures fail or redo.

I hope we will deserve to enter soon the civilized world.

Dear Tom,

There has been a dramatic decline in the aortoconary and also lower extremity bypass surgery. This is very clear both in the cardiac and vascular literature.

The primary vessels used for artocoronary bypass are arteries using of course always the LIMA that has the best patency rate amongst all bypass grafts

in the body. GSV is being used when arteries are not available or not enough. If the chance for using a GSV in the 1980s was around 3% now it is much less than that.

More importantly although a refluxing GSV may be preserved it is not known if such a vein has the same patency as a normal GSV. From histologic work we and others have performed on the saphenous wall of patients undergoing aortocoronary bypass there is moderate to severe fibrosis (due to age) in most patients undergoing such procedures. This is more common and more intense in patients with saphenous reflux.

In what the fibrosis of a saphenous vein is disputable or arterial by-pass, perhaps

Another issue is the diameter mismatch. For artocoranary bypass a vein of 3 to 4 ml is ideal and diameters up to 6mm are ok to use. Beyond this diameter the GSV is unlikely to be used.

Furthermore, we do ultrasound guided procedures on all patients and the results are better since there is no residual reflux and the recurrence rate is much less. With the endovenous techiques have resolved the neovasularization issue. Venous disease cannot be cure as with time new disease develops. We have now a long term follow-up in a good sample for over 5 years. The recurrence rate is over 20% as seen by detailed duplex ultrasound imaging in the standing position. Most of these patients are aymptomatic. Those who have symptoms in >95% of them the symptoms are milder compared to their original presentation.

A hemodynamic abnormality to some extent or completely may be corrected by CHIVA but the cosmetic outcome is not the same.

Why? Evidence? Rumor?? I spoke to few surgeons in Europe who tried CHIVA and had similar opinion Who are they? Were they expert enough? Where did they learn? . I also visited Paul and Sylvain in Nice. Both are great surgeons who perform ultrasound guided surgery at a high level and have a big interest on the pathophysiology and treatment of venous disease.

CHIVA requires less interventional work on the patient and even if provides the relief and cure that is claimed in the different publications (lets accept this as a fact for the time being) most of our patients will not be happy with such a procedure. I am not against the CHIVA and the people who promoting it. Most of them in fact are good friends. If in their hands this technique works well and their patients are happy it is fine with me. I have seen patients operated with this technique in Europe ( were? When?) and I had the opportunity to perform physical examination and ultrasound in a number of them. I was not very impressed by the outcome. Yet these patients I happened to see may be were failures or the procedure was not done properly, who knows. Anyway, I keep my mind open but in the mean time we are trying to do what we thing is best of our patients.

Have a nice day

Nicos

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Dear BB,

I'm not polite enough to hide my thoughts. I and we TRUST totally your honesty and your generous/great efforts you do for advances in Phlebology.

Best and warm regards

Claude

1. Zamboni P, Cisno C, Marchetti F, Mazza P, Fogato L, Carandina S, De Palma M, Liboni A. Minimally invasive surgical management of primary venous ulcers vs. compression treatment: a randomized clinical trial. *Eur J Vasc Endovasc Surg*. 2003 Apr;25(4):313-8.
2. Zamboni P, Cisno C, Marchetti F, Quaglio D, Mazza P, Liboni A. Reflux elimination without any ablation or disconnection of the saphenous vein. A haemodynamic model for venous surgery. *Eur J Vasc Endovasc Surg*. 2001 Apr;21(4):361-9.

**Dear BB,**

**I shall not go to IUP congress in Boston. I’m sorry and I beg your pardon, respect to my deep consideration for you and the huge efforts you are making. Fortunately, Massimo Cappelli ( D'artagnan) will represent me beside the 3 musketeers, Erika Mendoza, Stefano Ermini and Fausto Passariello. I look forward meeting you in Naples on November!**

**The reason is the surprising invitation by the UIP board limited to just a 10 minutes presentation of all the “venous shunts”, which cannot be less than “understandable” and deleterious for CHIVA communication. In addition, this “invitation” doesn’t include the subscription nor the travel expenses which, independently of the financial aspect, is against the usual rules. Acceptation would endorse this disrespect for the CHIVA school and it enormous and generous works regarding Phlebology.**

**Once again, forgive me.**

**Amitiés.**

**Claude**

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| |  | | --- | | **bblee** | | 16:18 (Il y a 15 heures)  https://mail.google.com/mail/u/0/images/cleardot.gif |  | **https://mail.google.com/mail/u/0/images/cleardot.gif**  **https://mail.google.com/mail/u/0/images/cleardot.gif** |
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Thanks to let me know your decision, Claude.

I would make a same decision if I were you. But I would like to explain on the background of Boston Congress NOT to make you feel humiliated.

Although I am still actively involved to the IUP, this particular IUP congress held in Boston is led by Am Coll Phleb(ACP) I have very very limited influence, NOT like Am Venous Forum(AVF), so that I wasn't interested to their program and naturally I did NOT know they allocated for 10 minutes to you for CHIVA/shunt presentation.

But, ten minutes-a limited time frame- allocation is NOT a big deal and no intention to humiliate you. It is rather a formality of the Congress Mark Meissner and Nicos Labropoulos took for the scientific program they chair together.

As you might know,  VEITH Symposium which represents the main stream of American vascular surgery took a new policy to allocate 6 minutes to 8 to 10 minutes in maximum to the most of topics, which all other congresses imitate including AVF and ACP.

Last December while I discuss on our Venous Hemodynamics with Mark Meissner and Andrew Nicolaides over the lunch during VEITH Symposium in New York, I recommended to Mark Meissner who is a Scientific Committee chair to organize the Boston scientific program, to consider to make a room for CHIVA to present two to three topics together to allocate 30 minutes altogether as a major topic.

So I don't know who else among CHIVA- perhaps Paolo or Fausto??- Nicos invited for other CHIVA related topics. But Mark Meissner has a limited role for this ACP-led Boston Congress though he is a Scientific Committee chair, and the majority of the final decision is being made by the ACP leadership.

Nevertheless, the mood is still pretty much hostile to you and other CHIVA leading members here on this side of the Atlantic. So you might have made a wise decision!

Regards,

BB

P.S. I would like to ask you to spare one evening for the dinner with me and my wife while in Naple so that we also could show our hospitality to you as well.

P.S. Ermini, Mendoza, and Passariello are good enough for CHIVA group for Boston Congress, no problem

**T**

**Thank you BB,**

**I don’t feel humiliated, but I don’t want to endorse what I feel negative for the diffusion of knowledge. The congresses are like Procuste’s bed. They fit only to short “messages” ( for example, I could easily present “the intermittent obstruction of the thoracic duct end “ quickly understandable despite so far unknown because it’s the matter of “blocked or not blocked” and 2 Images). But the bed is too short for concepts that are at the same time “new” and “complex” and need background as physics, unfamiliar to the phlebologists. Accepting such a bed, is killing CHIVA bcause we must behead it. The hostility against us is not only on your side of the Atlantic, but “home” also. I don’t feel angry about these enemies as persons who I pity for their suffering. I see in their eyes a kind of distress, for not understanding at the first time, that make them run away and gossip in the back stage against us ( charlatans, sect, religion) , deny the evidences theoretical and material we provide, while few others take time and become friends when , freed from their “normal prejudices” they understand that the matter is “easy” to digest for who takes time to chew it slowly. So, my aggressive behavior is not against them, but to defend the patients, and who want to learn/understand some.**

**Thank you for your invitation at dinner!**

**Amitiés**

About Jargon, I would like to know if it is a grammar/syntax problem of some terms as "Vicarious, Diviated, Shunts..." despite they exist in the dictionary. For the grammar syntax, the book "Principles of Hemodynamics" was rewritten by an english MD. I do agree to correct whatever of CHIVA in English, if the significance remains correct. Who could help us? As non English speakers, we make a huge effort to understand , speak and write in this language. Perhaps the English speakers could be a little grateful and help us F

About Jargon, I would like to know if it is a grammar/syntax problem of some terms as "Vicarious, Diviated, Shunts..." despite they exist in the dictionary. For the grammar syntax, the book "Principles of Hemodynamics" was rewritten by an english MD. I do agree to correct whatever of CHIVA in English, if the significance remains correct. Who could help us? As non English speakers, we make a huge effort to understand , speak and write in this language. Perhaps the English speakers could be a little grateful and help us instead of blaming.

**Dear Puskas,**

**Is it possible to treat easily deep venous incompetence with recurrent ulcers despite correct compression and dressing ? Yes on 2 conditions: 1- presence of deep closed shunts , i.e competents collaterals that could allow ligation of the incompetent vein ( double superficial femoral vein where the incompetent may be divided because the other one is competent, supressing so the reflux and draining properly through the competent or superficial femoral vein division if the deep femoral vein is competent and connects the popliteal vein to the femoral vein, or division of the posterior tibial vein at a level were the skin is healthy.2- An accurate Doppler assessment in order to draw a correct mapping. SEE ATTACHED FILE**

**Deep CHIVA is not possible in absence of deep closed shunt . Here, valve construction/graft is necessary and Oscar Maletti is an excellent address.**

**We perform this technique since more than 2 decades and so far only in recurrent resistant ulcers in around 10 patients, with satisfactory results (ulcer healing). If you are interested, let me know .**

**We performed these operations in 2 days recovery because of the anticoagulation**

**All the best**

**Claude**

**Dear Raj**

**I have read the reprint.**

You write: Increasing the extravenous pressure did not decrease TMP. intravenous pressure simply rose to equal the external pressure applied with the result there was no change in TMP. The increase was apparently due to conversion of velocity to pressure energy per Bernouli

**Claude: In this experimentation, the Pressurizing reservoir (11)  measures ONLY variations of the pressure/volume of the Penrose and NOT external pressure. The external pressure is given by the volume/weight of the Pressurizing reservoir, that doesn’t vary in this experimentation. So, the measure of the TMP by the difference between Penrose pressure and the pressure shown by Pressurizing reservoir, is wrong and will not change with the changes of the Penrose pressure. . In fact, the TMP is the difference between  the Penrose pressure and the pressure provided by the volume/weight of Pressurizing reservoir. When the  downstream resistance is low , the TMP decreases. It is the case when the compression at the leg is applied in normal or incompetent veins in absence of downstream high resistance. In case of downstream high resistance, before compression, the TMP is high because the IV pressure is high  due to accumulation  of Residual Pressure. Compression will increase the external pressure and will decrease the TMP in reverse proportion to the downstream resistance (that increases the lateral pressure).**

**This is a typical case of mistakes that occur when the “physical initial data”  are not clearly defined.**

**I hope I have been understandable despite my broken English. Tell me...**

**Amitiés.**

Dear Claude,

I appreciate your deep fluid mechanical knowledge and value your opinion highly to understand things myself (an amateur). In this experiment, the pressure within the penrose was measured but the energy input fed by gravity from a reservoir of fixed height remained constant. However, the pressure (outside the penrose) inside the pressurizing reservoir was progressively changed by changing the height of the fluid reservoir that pressurized it. I was expecting to see compression of the penrose with increase in the pressure outside the penrose but it did not happen. The caliber of the penrose remained the same but the velocity of flow inside the penrose slowed greatly while the pressure inside the penrose rose by an amount corresponding to the calculated increase in the pressurizing reservoir outside the penrose. The inference was that the pressure inside the penrose rose from conversion of velocity to pressure. We did not have velocimeters but timed flow volumes were used as a measure of velocity since the tubing size from the gravity feed remained the same.

What do you think?

Many regards and appreciation of your intellectual vigor in explaining things.

Raj

**From:** Claude FRANCESCHI [mailto:[claude.franceschi@wanadoo.fr](mailto:claude.franceschi@wanadoo.fr" \t "_blank)]   
**Sent:** Monday, August 19, 2013 10:01 AM  
**To:** seshadri raju  
**Subject:** re: Reprint

**Dear Raj,**

**Here is my comment :**

Raj:In this experiment, the pressure within the penrose was measured but the energy input fed by gravity from a reservoir of fixed height remained constant. However, the pressure (outside the penrose) inside the pressurizing reservoir was progressively changed by changing the height of the fluid reservoir that pressurized it

**Claude:*I guess, you didn’t add liquid outside the penrose but increasing the resistance to the penrose flow , the penrose liquid volume/pressure increased, increasing at the same time the level of the external liquid BUT NOT ITS pressure***. ***You would be right if you have added extra penrose liquid …but in that case the TMP would have decreased with this external pressure and increase with penrose Lateral pressure that increases with the resistance to the penrose flow.***

Raj: I was expecting to see compression of the penrose with increase in the pressure outside the penrose but it did not happen. The caliber of the penrose remained the same but the velocity of flow inside the penrose slowed greatly while the pressure inside the penrose rose by an amount corresponding to the calculated increase in the pressurizing reservoir outside the penrose. The inference was that the pressure inside the penrose rose from conversion of velocity to pressure

**Claude:** ***I agree*, this is a basic of Bernouilli law. .** We did not have velocimeters but timed flow volumes were used as a measure of velocity since the tubing size from the gravity feed remained the same.

**Claude :you can theoretically mesure the velocity with the Bernouilli equation, if you know the variation of lateral (static)pressure and the mass of the liquid.**

**I hope I replied correctly… The issue of hemodynamics is that physicists doesn’t know enough about medicine and physicians not enough about physics. Surely, I’m not a good English speaker and I may be a right reason for my “ Jargon”. Surely also, Jargon is the easy name we give to what we don’t understand ( my mother tongue is French ….but many French colleagues repulse my explanations because “jargon” or because they are angry for understanding not? . Perhaps it is their problem, not mine, since I have no understanding nor jargon problems with who understands or wants to ). I’m an amateur who made the efforts to understand as a professional …. I appreciate your curiosity and open mind and I'm delighted to learn from each other.**

**Let me know your opinion.**

**Amicalement**

**Claude**

Dear Claude,

The pressurizing chamber was filled with water (flow medium in the penrose also) and connected to a gravity fed tank. The pressure in the chamber was varied by altering the height of the pressurizing tank. The key finding was that increasing the outside pressure *did not*decrease the TMP. It remained unaltered because the lateral pressure inside the penrose increased by an equivalent amount to neutralize the increased pressure outside. So the penrose did not compress, it maintained the same caliber as before.

I find your English quite descriptive and indeed has a musical quality to it.  You are a great mind and perhaps others find it intimidating. For myself, I am glad to receive your insights.

Regards,

Raj

Raj

 Thanks for your nice  pictures.

1- Are membranes primary  or secondary to the compression?  Is the spinal curvature the cause of compression, then membranes? I don't know and I'm not used to believe what I don't know.

2- Decades ago we divided ( Pr Cormier department  Paris) the left comon iliac artery and transposed it on the   the right iliac artery in few patients without substantial clinical outcomes.

3- The presence of collateral veins in venography disappeared  when 45° seated  in most cases , attesting the postural compression more determinent than membranes.

4- The absence of venous hypertension in supine attests the efficiency of the natural compensatory of the collaterals..at least at rest. A pressure measurement when walking should check the collateral efficiency when the flow increases ( We didn't perform it...but why not to study it?). This could be the most logical/rational way to select the patients eligible for specific treatment.

2013/9/9 miguel amore <[miguelangelamore@hotmail.com](mailto:miguelangelamore@hotmail.com)>

Dear Prof Franceschi, My name is Migue Amore, Im a vascular surgeons and I work at the department of phlebology and Lymphology  at the Central Military Hospital of Buenos Aires, and at the  Anatomy Department of Buenos Aires University.

Regarding the May Thurner syndrome or Cockett syndrome,  we prefer the term: left common iliac vein compression, which is related to the clinical manifestations of compression and not  on the iliac vein thrombosis.

In our experience the most characteristic  in this syndrome is  the collateral circulation (presacral and ascending lumbar veins), Fact that evidence the  venous hypertension  by the compression and  the presence of membranes in the lumen of the vein.

Mebranes presence does not change with the positions taken by the patient supine or seated 45 °. Compression maybe?

In many of our patients there was not much change in the pressure gradient.

but it evidenced the presence of collateral circulation in all of them.

In all our patients realize the iliocavography in different positions to rule out functional compressions.

The question is: if all humans have the crossing, why only a few patients have membranes and symptomatologies of compression. Perhaps the pathology of spinal curvature play a role in this syndrome or an embryology defect? What do you think?.

PS. I have attached some images of my anatomical dissections and its correlation with some of my patients.

Claude

Fantastic! Thanks!

Though my understanding of French language is very limited, I was able to retrieve most of the essence along the French concept you nicely reviewed for us. Are you going to bring this to Hemodyn Meeting in Napoli? It would be able to give us different aspect of the CVM we would love to hear more.

Indeed, I was quite impressed to learn French concept, though you said it belongs to '70, seemed to have quite advanced, then, comparing to our traditional old- some calls it 'continental' - concept; it is rather very close to the concept we established on the CVM along the era of 1988 when world  experts in this unique field organized the world consensus workshop held in Hamburg. Based on their consensus, two new classifications were established and your PPT/summary seems to fit just in between two different classifications.

Nevertheless, we no longer use the term of Angioma in order to avoid unnecessary confusion with 'hemangioma' which is NOT a vascular malformation but vascular tumor- see the attached-, and instead we adopted/borrowed old embryological term, 'extratruncular' for such lesion with distinctive morphological appearance induced by mesenchymal cell characteristics, for the Hamburg Classification-see the attached-. Together with 'truncular' lesion which represents the defects originated from the 'late' stage of embryogenesis, this extratruncular lesion became the base of Hamburg Classification for us.

Another interesting fact I found in your PPT is French school used the term 'capillary' for the 'extratruncular' lesion. Such lesion is the outcome of defective development along early, reticular stage of embryogenesis so that it remained *dysplastic network* but NOT a true normally developed and matured capillary system. So we avoid to use the term of 'capillary' except for genuine 'capillary malformation' known as port wine stain, etc.

I also amused to see you use the term, FAV(=AV fistula) to both capillary/extratruncular and troncular/truncular AVM, which the ISSVA group should learn- they classified the AV malformation to AVM and AVF despite all the AVM lesions, either truncular or extratruncular, are 'fistulous' condition between the artery and vein. No fistula, no AVM. It has become one of the reasons why I gave up further argument with ISSVA leadership umpteen years ago and divorced with ISSVA to organize a new camp of Hamburg Classification aschool.

Anyhow, it is amazing to find out that French school had quite advanced concept already in '70 era!

Thanks,

BB

P.S. ISSVA Classification embraced all the name-based eponyms as well as syndrome-based classification against the Hamburg classification. But we belonging to Stefan Belov's original Hamburg Classification respectfully disagree with them and use modified Hamburg Classification now.

P.S. One small correction on your PPT, if you would: Kasabach–Merritt syndrome (KMS) based on the previous concept, which is now found to be different from the coagulopathy among the VM patient which is secondary to 'localized intravascular coagulopathy' (LIC). I will include a part of my chapter in 'Clinical Handbook for the Management of Antithrombotic and Thrombolytic Therapy in Venous Thromboembolism' edited by Univ Minn group (Gundu Rao and Jawed Fareed et al) which will soon be available.

10-3. Coagulopathy of the VM    10-3-1. General Overview

Confusion in the nomenclature and classification of CVMs and the hemangioma, has hindered the appropriate understanding of the coagulopathy associated with these conditions. The coagulation abnormality occurring in the VMs is due to the 'localized intravascular coagulopathy' (LIC) (5-8, 147-150) but often misdiagnosed as Kasabach–Merritt syndrome (KMS) (23-26, 151, 152).

KMS is a hallmark of coagulopathy belonging to the hemangioma/vascular tumor.  Despite marked differences in clinical features, pathophysiology and its management of both conditions as mentioned

above, confusion still remains a threat to proper treatment of the VM (24). LIC associated with the VM lesion is a distinctive, lifelong coagulopathy.  Blood stagnation within the VM lesion results in thrombosis leading to phlebolith formation ( 6, 97). The finding of palpable phleboliths and an elevated D-dimer level, remains a hallmark of LIC in VM patients. In contrast, KMS is initiated by *platelet trapping within a vascular tumor* that develops in infancy.

10-6.  Kasabach-Merritt Syndrome     10-6-1.  General Overview

Kasabach-Merritt syndrome (KMS) is a rare, life-threatening condition involving immature blood vessels of vascular tumors that form a large growth and interfere with coagulation. KMS is also known as hemangioma thrombocytopenia syndrome (HTS) and more recently has been referred to as the Kasabach-Merritt phenomenon (KMP). Vascular tumors causing KMS can be found in the trunk, upper and lower extremities, retroperitoneum, and in the cervical and facial areas (153).

Dear BB,

The ppt was the classification I proposed in the seventies , based on my findings and the consequent concepts I designed according to hemodynamics , principally the pencil CW Doppler data ( DUS was not yet born). The French vascular community didn’t follow me, excepted Pr JM Cormier ( a clever opened minded pioneer) and who was the chief department of vascular surgery St Joseph hospital Paris where I led the vascular diagnosis. Then, he retired, and I carried on with Dr Claude Laurian. I kept away from of the French school most represented by radiologists and led by Pr Merlan, because I didn’t agree with their “systematic embolisation of AVM. It was quite impossible to publish any of these concepts because not accepted ( too complicated? Not in accordance with the current consensus? Bullshit?) I was happy to see the international evolution, particularly led by you, that confirmed most of my classification. Then, I had to face once again misunderstandings, false rumors and silent anger of the vascular community when I proposed hemodynamic concepts that resulted in CHIVA. At the moment of the PPT, the updated informations about Kasabach-Merritt Syndrome didn’t yet exist. On the other hand, the struggle about names hides the real issue, i.e the concepts they address to. It remembers me the endless and improductive Byzantin discussions. Later, I had the opportunity to publish in French some papers as ATTACHED:

-Les malformations vasculaires congénitales in Encyclopédie pratique d'échotomographie et de Doppler vasculaire. Bristol-Meyers Squibb 1995 C. FRANCESCHI C comprehensive text and figures likely usefull even for non-french speakers and an other

-Traitement chirurgical des malformations vasculaires des membres encyclopedie medico-chirurgicale, 2004:(68),43-260

As you can see, these works were not read/accepted in France, out of our Saint Joseph’s team. I let you find the reason why… In addition, the French community NEVER invite me to publish nor to speak in their meetings, despite I tought the first wave of angiologists and some surgeons. Nevertheless, I don’t complain for myself, since I had the pleasure to develop my knowledge and share it with some colleagues...and now with you. Despite my reluctance to show off, I have to state some truth and facts related to my work.

Warm regards

Claude

LAURIAN, C., FRANCESCHI, C., HERBRETEAU,D., ENJOLRAS,O.:   
TRAITEMENT CHIRURGICAL DES MALFORMATIONS VASCULAIRES DES MEMBRES ENCYCLOPEDIE MEDICO-CHIRURGICALE, 2004:(68),43-260

Dear Olle Nelzen,

Thank you for this interesting information. Nevertheless, we must make sure that that Lena Blomgren and Oriol Pares RCTs are comparable in terms of method and material. Indeed, at the first glance, in the Lena’s study Duplex was performed in one group and not in the other, while in Oriol’s, all the patients were diagnosed by Duplex performed by the same specialists prior stripping and CHIVA (2 groups CHIVA and Stripping were echo-marked on the skin before the procedures while a third group was clinically marked). Such bias could make the comparison between the 2 sudies not acceptable. For the reason, I suggest you mail us the PDF of Lena’s RCT while I send you the Oriol’s (**Parés JO, Juan J, Tellez R, Mata A, Moreno C, Quer FX,et l.Varicose vein surgery: stripping versus the CHIVA Method: a randomized controlled trial. Annals of Surgery 2010;251(4):624–31)** accompanied by a Cochrane review **(Bellmunt-Montoya S, Escribano JM, Dilme J, Martinez-Zapata MJ. CHIVA method for the treatment of chronic venous insufficiency. Cochrane Database of Systematic Reviews 2012 , Issue 2 . Art. No.: CD009648. DOI:10.1002/14651858.CD009648)**, in order to make an accurate analysis of both studies and see the comparison is pertinent.

Duplex method

As Joe underlined, the Duplex quality is very variable…Find attached a PDF of a presentation I did in Hemodyn 2012.

Dear Claude,

Thank you for your good work throughout the years. I and others may not agree in some areas with you but this does not mean

that we do not recognize and appreciate your work. It is unfortunate that your work was not accessible for most people as it was not

published in well known peer reviewed journals but there are some of us who look deeper. I can assure you that you have done

nice work that often was ahead of its time and therefore not well perceived by many. We are living in world that is becoming digital

and therefore, as you have seen at least through the vasculab many people have access to your views and works. So continue your good

work and enjoy!

Have a nice weekend

Nicos

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**From:** claude franceschi <[claude.franceschi@gmail.com](mailto:claude.franceschi@gmail.com)>  
**To:** "[vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com)" <[vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com)>   
**Sent:** Saturday, September 28, 2013 7:55 AM  
**Subject:** Re: [vasculab] VM and AVM

Dear BB,

The ppt was the classification I proposed in the seventies , based on my findings and the consequent concepts I designed according to hemodynamics , principally the pencil CW Doppler data ( DUS was not yet born). The French vascular community didn’t follow me, excepted Pr JM Cormier ( a clever opened minded pioneer) and who was the chief department of vascular surgery St Joseph hospital Paris where I led the vascular diagnosis. Then, he retired, and I carried on with Dr Claude Laurian. I kept away from of the French school most represented by radiologists and led by Pr Merlan, because I didn’t agree with their “systematic embolisation of AVM. It was quite impossible to publish any of these concepts because not accepted ( too complicated? Not in accordance with the current consensus? Bullshit?) I was happy to see the international evolution, particularly led by you, that confirmed most of my classification. Then, I had to face once again misunderstandings, false rumors and silent anger of the vascular community when I proposed hemodynamic concepts that resulted in CHIVA. At the moment of the PPT, the updated informations about Kasabach-Merritt Syndrome didn’t yet exist. On the other hand, the struggle about names hides the real issue, i.e the concepts they address to. It remembers me the endless and improductive Byzantin discussions. Later, I had the opportunity to publish in French some papers as ATTACHED:

-Les malformations vasculaires congénitales in Encyclopédie pratique d'échotomographie et de Doppler vasculaire. Bristol-Meyers Squibb 1995 C. FRANCESCHI C comprehensive text and figures likely usefull even for non-french speakers  and an other

-Traitement chirurgical des malformations vasculaires des membres encyclopedie medico-chirurgicale, 2004:(68),43-260

As you can see, these works were not read/accepted in France, out of our Saint Joseph’s team. I let you find the reason why… In addition, the French community NEVER invite me to publish nor to speak in their meetings, despite I tought  the first wave of angiologists and some surgeons. Nevertheless, I don’t complain for myself, since I had the pleasure to develop my knowledge and share it with some colleagues...and now with you. Despite my reluctance to show off, I have to state some truth and facts related to my work.

Warm regards

Claude

Claude Franceschi

2013/9/27 bblee <[bblee38@comcast.net](mailto:bblee38@comcast.net)>

Claude

Fantastic! Thanks!

Though my understanding of French language is very limited, I was able to retrieve most of the essence along the French concept you nicely reviewed for us. Are you going to bring this to Hemodyn Meeting in Napoli? It would be able to give us different aspect of the CVM we would love to hear more.

Indeed, I was quite impressed to learn French concept, though you said it belongs to '70, seemed to have quite advanced, then, comparing to our traditional old- some calls it 'continental' - concept; it is rather very close to the concept we established on the CVM along the era of 1988 when world  experts in this unique field organized the world consensus workshop held in Hamburg. Based on their consensus, two new classifications were established and your PPT/summary seems to fit just in between two different classifications.

Nevertheless, we no longer use the term of Angioma in order to avoid unnecessary confusion with 'hemangioma' which is NOT a vascular malformation but vascular tumor- see the attached-, and instead we adopted/borrowed old embryological term, 'extratruncular' for such lesion with distinctive morphological appearance induced by mesenchymal cell characteristics, for the Hamburg Classification-see the attached-. Together with 'truncular' lesion which represents the defects originated from the 'late' stage of embryogenesis, this extratruncular lesion became the base of Hamburg Classification for us.

Another interesting fact I found in your PPT is French school used the term 'capillary' for the 'extratruncular' lesion. Such lesion is the outcome of defective development along early, reticular stage of embryogenesis so that it remained*dysplastic network* but NOT a true normally developed and matured capillary system. So we avoid to use the term of 'capillary' except for genuine 'capillary malformation' known as port wine stain, etc.

I also amused to see you use the term, FAV(=AV fistula) to both capillary/extratruncular and troncular/truncular AVM, which the ISSVA group should learn- they classified the AV malformation to AVM and AVF despite all the AVM lesions, either truncular or extratruncular, are 'fistulous' condition between the artery and vein. No fistula, no AVM. It has become one of the reasons why I gave up further argument with ISSVA leadership umpteen years ago and divorced with ISSVA to organize a new camp of Hamburg Classification aschool.

Anyhow, it is amazing to find out that French school had quite advanced concept already in '70 era!

Thanks,

BB

P.S. ISSVA Classification embraced all the name-based eponyms as well as syndrome-based classification against the Hamburg classification. But we belonging to Stefan Belov's original Hamburg Classification respectfully disagree with them and use modified Hamburg Classification now.

P.S. One small correction on your PPT, if you would: Kasabach–Merritt syndrome (KMS) based on the previous concept, which is now found to be different from the coagulopathy among the VM patient which is secondary to 'localized intravascular coagulopathy' (LIC). I will include a part of my chapter in 'Clinical Handbook for the Management of Antithrombotic and Thrombolytic Therapy in Venous Thromboembolism' edited by Univ Minn group (Gundu Rao and Jawed Fareed et al) which will soon be available.

10-3. Coagulopathy of the VM    10-3-1. General Overview

Confusion in the nomenclature and classification of CVMs and the hemangioma, has hindered the appropriate understanding of the coagulopathy associated with these conditions. The coagulation abnormality occurring in the VMs is due to the 'localized intravascular coagulopathy' (LIC) (5-8, 147-150) but often misdiagnosed as Kasabach–Merritt syndrome (KMS) (23-26, 151, 152).

KMS is a hallmark of coagulopathy belonging to the hemangioma/vascular tumor.  Despite marked differences in clinical features, pathophysiology and its management of both conditions as mentioned

above, confusion still remains a threat to proper treatment of the VM (24). LIC associated with the VM lesion is a distinctive, lifelong coagulopathy.  Blood stagnation within the VM lesion results in thrombosis leading to phlebolith formation ( 6, 97). The finding of palpable phleboliths and an elevated D-dimer level, remains a hallmark of LIC in VM patients. In contrast, KMS is initiated by *platelet trapping within a vascular tumor* that develops in infancy.

10-6.  Kasabach-Merritt Syndrome     10-6-1.  General Overview

Kasabach-Merritt syndrome (KMS) is a rare, life-threatening condition involving immature blood vessels of vascular tumors that form a large growth and interfere with coagulation. KMS is also known as hemangioma thrombocytopenia syndrome (HTS) and more recently has been referred to as the Kasabach-Merritt phenomenon (KMP). Vascular tumors causing KMS can be found in the trunk, upper and lower extremities, retroperitoneum, and in the cervical and facial areas (153).

**From:** [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com) [mailto:[vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com" \t "_blank)] **On Behalf Of**claude franceschi  
**Sent:** Thursday, September 26, 2013 5:39 AM  
**To:** [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com)  
**Subject:** [vasculab] VM and AVM

Dera BB

See attached the PPt I made in the seventies where I proposed an hemodynnamic  classification and therapeutic indication.

About AVM, I totally agree with you. In Paris, in the seventies we performed embolisations with dura....Doppler guided to avoid necrosis in healthy tissues....with fantastic immediate outcomes (normal angiogram) and awfull mid term recurrence. WE STOPPED this procedure. Later, the radiologists ( french school) endeavoured AVM treatments based on embolisation, and they asked me to participate. I refused.  AVM is still a challenge where the "angiogenetic potential is not yet controlled.Large surgical excision , when possible, is so far the only  efficient choice. Embolisation in non-operable patients when they suffer bleeding or heart failure. In some selected cases as finger distal AVM, compresion may be very usefull .

About MV, echoguided surgery i accordance with hemodynamic strategy, when possible and indicated ( pain, knee compromission,  or cosmetic concern). Embolisation of refluxing malfomative pelvic veins. No foam in the large majority of the cases ( we operate patients who suffered recurrence and more pain after foam).

I stop here and I look forwards exchanging more with you in  Naples Hemodyn November 2013.

Warm regards.

Claude Franceschi

2013/9/26 bblee <[bblee38@comcast.net](mailto:bblee38@comcast.net)>

Thanks, Claude.

My point is the best way to reach to the goal shortening the learning curve is the learning from other's mistake/mishap. That is the reason why American medicine/surgery exceeded its counterpart across the Atlantic - excuse my arrogant(?) description/attitude-because American concept is pretty much based on the pragmatism in every possible way. So I love weekly M & M (mortality & morbidity) meeting how others also screw up like I do, not only making me feel better but learn how to avoid same mistake. I don't care the statistics because clinical medicine is an art and not a 100% science. So I always appreciate the colleagues to share/show their dirty laundry to let others learn.

Neverthless, 'deep CHIVA' is certainly interesting approach to stimulate the traditional approach to giving oneself second look to identify/confirm where oneself is standing now.

Regards,

BB

P.S. Embolization to the AVM and VM are two different matters with entirely different prognosis; When you embolize the AVM, you  practically burn down the bridge to cross later in contrary to the VM. Further, the embolization of the feeding artery -French school used to lead this profound mistake based on deficient knowledge for many decades through last century resulting in tremendous harm to the patient- only stimulates this embryonic tissue remnants with mesenchymal cell characteristics to go wild with neovascular recruitment to cause rapid growth/recurrence. So we are now clever enough to imply discriminating use of various embolo-sclerotherapy with/without surgical excision combined.

**From:** [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com) [mailto:[vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com" \t "_blank)] **On Behalf Of**claude franceschi  
**Sent:** Wednesday, September 25, 2013 3:01 AM

**To:** [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com)  
**Subject:** Re: [vasculab] Deep vein valve reconstruction

Dear BB,

I don't want to undisclose the bad results....because they are a good reason to stop a bad treatment. For example, the good primary results of embolization in AVM and their awfull recurrences some months later made me stop embolization many decades ago and prefer surgery when possible and embolization when surgery is not feasible but mandatory for heart failure or bleeding. For the same reason, I don't like foam in VM and prefer surgery when feasible and the case really need a treatment . The results of deep CHIVA, a choguided surgery feasible under local anesthesia in most cases ,  are all positive in the selected cases , i.e where and when it is indicated as I wrote in my previous mails. Few cases even if all successful are not statistically valuable, but they open a way for knowledge/ therapy improvement. CHIVA in those cases is easy for who knows enough about venous hemodynamics and DUS....look at the file I attached, showing the CHIVA strategy in deep venous insufficiency,  in my previous mail. Monoscup or Maletti's  valve construction could an optional when CHIVA is not feasible.

BW

Claude.

Claude Franceschi

2013/9/24 bblee <[bblee38@comcast.net](mailto:bblee38@comcast.net)>

Claude  
  
I understand your reluctance and clinical cases to imply this new approach  
wouldn't be that many in comparison to ordinary varicose vein.  
  
But even 10 cases will be good enough to show its interim results if the  
data can deliver an objective evidence. It would be rather much better if  
you have any failed/non-responding cases to compare those with  
good/satisfactory outcome. We all learn from other's dirty laundry!  
  
I still vividly remember that more than 20 years ago when there was an  
excellent chance to Genoa group to share their excellent outcome of  
lymphatic reconstruction with others/U.S. colleagues, they made a fatal(?)  
mistake to disclose only the best results but evaded the chance to share  
their dirty laundry so that it generated a biased opinion even among  
potential supporters with a suspicion to hide the facts. This unfair  
suspicion provided a biased opinion with prejudice they are still paying  
for.  
  
Regards,

BB  
  
From: [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com) [mailto:[vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com" \t "_blank)] On Behalf  
Of claude franceschi

Sent: Tuesday, September 24, 2013 3:53 AM

To: [vasculab@yahoogroups.com](mailto:vasculab@yahoogroups.com)  
Subject: Re: [vasculab] Deep vein valve reconstruction

Dear BB, the clinical data are limited to few patients ( not more than 10)  
though we see many patients and performed thousands of superficial CHIVA.  
Why? Because we perform deep CHIVA only in case of resistant Ulcer and in  
presence of deep shunt. All the few operated patients were cured. On the  
other hand, I think that many ulcers are diagnosed to be due to deep  
incompetence just because of large perforators below the knee though they  
are most of the time re-entries of important superficial shunts. Sometimes,  
those large perforators are associated with deep venous reflux, but they  
don't show a diastolic reflux because they are connected to a competent deep  
compartement ( e.g competent posterior tibial while the popliteal refluxes  
in other veins of the calf). In addition and according to my experience, , a  
resistant ulcer is not necessarily due to deep incompetence but may be due  
to a superficial inadequate treatment Unfortunately, the number of our deep  
CHIVA is not sufficient for a publication, but its rational should deserve  
it. but the current custom for publication is not deductive science which  
was/is the basis of the great discoveries along the history, i.e  
concept/model based that I used in all my research. Today, the gold standard  
( in medeine) seems to be only inductive.  
  
BW  
  
Claude

Claude Franceschi

Many thanks for sharing your Power Point animations with us. As in previous messages, here and elsewhere, I do respect your attempts to understand this complex area and am looking forwards to discussing these with you at your course.

However, as you have put the animations up, there are many points that I feel are inaccurate - and I hope we will discuss these in Cremona.

However, relevant to this discussion and the use of TRLOP to close incompetent perforators, in the whole of your presentation, you show blood going INWARDS through enlarged perforators as re-entry points UNLESS there is an obstruction. But inwards flow in a perforator is the normal direction ie: NOT incompetent.

The times you do show OUTWARDS flow ie: incompetence in the perforator, there is an obstruction in the deep veins proximal to the perforator.

However in real life, we have hundreds of patients with no deep vein obstruction, with outward flow in the incompetent perforators, where either there is no reflux in the truncal vein or this has been closed - yet the incompetent perforator still has outward flow during the active phase ("systole" in your terms) leading into varicose veins, thread veins or skin changes.

Closing the perforator in these people cures the problem.

But you ignore this in your presentation.

Hence your explanation, eloquent and well presented as it is, doesn't tally with the clinical cases that I see and treat in a great number of patients.

My interest is not to argue nor to score points - merely to understand the venous system so we can all learn the optimal way to treat our patients.

As such, I do believe that you are overlooking a very important pattern and down grading the importance of perforator vein reflux in some patients.

The other areas that I have problems with in your animations will be great to talk through with you in December.

Very best wishes,

\_\_.\_,\_.\_\_\_

Cher Mark,

I’m afraid, there is a misunderstanding. A systolic perforator outwards flow occurs if 2 conditions are fulfilled, a valve incompetence of the perforator and a low resistance in the superficial vein it connects. The more the resistances in the deep veins , the more the outward flow. This complies with the Ohm’s laws (parallel resistances). A diastolic perforator inwards flow is a normal direction that complies with the physiological drainage hierarchy , whatever is large or narrow caliber. The anomaly relates not to the direction (normal) but to the overloading pressure and flow caused by the shunts (closed or open) , where they play the role of re-entry. Some times, the same perforator refluxes outwards during the systole while it flows inwards during the diastole. . In that case, the systolic reflux is called “compensated” when less important than the diastolic inflow. It is the case in many closed shunts re-entries. We never close them in that case, in order to respect the drainage while the escape point is disconnected and the cosmetic results are excellent. The solely systolic refluxing perforator in a “normal” deep-superficial venous system , occurs frequently and is “asymptomatic” ….except in some sportsmen, where the venous flows during sport is very high ( volume and pressure) and where the perforators closure leads to further catastrophic recurrences. This is the observation made by the CHIVA group over the world and myself, regarding thousands of patients since 30 years. The animation is necessarily schematic and not comprehensive , nevertheless in accordance with the CHIVA theoretical basis. .

COINCIDENCE!

I just saw yesterday a woman (51 years old) who presented the same configuration as you patient ( except she didn’t have deep venous incompetence at the right lower limb, but only GSV incompetence). We operated her 16 years ago ( she was 35) and no recurrence until 3 weeks ago……

What did we do 16 years ago and why a recurrence 16 years later?

We performed successively a superficial CHIVA on the GSV and 2 staged Deep CHIVA on the Superficial vein and on the Posterior tibial vein ( successively in 1 year of time because the first 2 steps reduced dramatically the ulcer but a 1cm² ulceration remained, definitively cured by the third step 3 months later). Then, elastic stockings …light and elegant because she worked as barmaid in a “chic” Parisian Night bar. The recurrence? No edema nor swelling, soft skin, but a 1/2cm² ulcer. The cause? The Deep CHIVA disconnection are not recanalised but the superficial Femoral vein division is “by-passed “ by a collateral small incompetent vein. No recurrence of the GSV closed shunt but incompetent column of the GSV is prolonged now down to the ulcer.

What shall we do? A 5 minutes under local anesthesia fractionation of the GSV just below the knee ( so distant from the ulcer) will be sufficient to heal it again for …16 years more?

For your patient, I suggest you begin with GSV CHIVA, and go on with deep CHIVA if 2 months later the ulcer is not healed. ( If you wish, I could send you more informations about deep CHIVA procedure , assessment and treatment).

Coincidence? We did other deep CHIVA but not in such topographic/hemodynamic configuration.

Amitiés

I agree with Michèle Cazaubon consideration about the deep venous thrombosis etiology to be checked.

reflux occurred

Dear Dimitru,

Why not OB CHIVA…but we don’t know at this time how long the GSV closure lasts.

Deep CHIVA: The Superficial femoral vein division ( with at least 4 cms resection) under DUS skin Marking only when it is part of a deep closed shunt i.e

when refluxing during the diastole and its collateral ( double Superficial femoral Vein or Deep femoral vein connects to the popliteal vein , i.e when their antegrade flow is evoked by the calf squeezing or Paranà) . Deep CHIVA not feasible in absence of deep closed shunt, i.e when all the thigh deep vein are incompetent.

Deep CHIVA at the Post Tibia vein consists of 3 cms resection of the vein ( or the 2 PTV ) above the Ulcer , where the skin is heathy.

See 2 attached files: diagnosis and strategy

BW

shocking!

Dear BB,

Novelties are provocative ....because they burst the current paradigm. CHIVA concepts were/are shocking : while the paradigm of treatment of the varicose veins was destruction , CHIVA proposes conservation. While the paradigm was valve respect, CHIVA proposes devalvulation in Shunts III. While the paradigm was prevention of deep veins obstruction, deep CHIVA proposes deep veins division in deep closed shunts. These heretic concepts comply with a model which is coherent with physics and physiology assessment of the venous insufficiency. A severe misunderstanding would be defining CHIVA as disconnection and devalvulation of the GSV, ligation/division of the deep veins. Because these procedures are beneficial or deleterious according to the hemodynamic configuration of each venous insufficiency. That's the reason why an accurate diagnosis/topographic and hemodynamic cartography , performed and analysed by through a consistent knowledge of venous hemodynamics is mandatory for CHIVA. CHIVA for Mummies, would lead to catastrophe ....Fortunately, phlebologists are not mummies but the professionals who are supposed to make the necessary effort to fully understand the hemodynamics venous pathophysiology. BTW, deep CHIVA is not published ( while presnted by Jordi maeso some years ago at a French congress of the College francais de Pathologie vasculaire). But its is "logical" in presence of deep closed shunts and Maeso's results as mine ( around ten patients along decades...) were all positive and no worsening).

Warm regards

Dear all,

KTS is a peculiar anatomic and hemodynamic configuration of the venous system among others due to embryologic trouble. Nevertheless it obeys the venous hemodynamics laws , as any other venous disease. The persistent marginal vein is not specific nor necessary for KTS. ATTACHED PPT.

The Klippel-Trenaunay-Weber Syndrome was described as

- a metameric venous malformation limited to one lower limb,

- without ArteroVenous Fistule

- sometimes combined with lymphatic malformation,

- consisting of

-superficial varicose veins,

-deep veins agenesia, usually popliteal and/or superficial femoral vein

-combined with

- limb hypertrophy particularly skeletal and

- superficial capillary angioma.

The persistent marginal vein not specific nor necessary for KTS.

Such syndrome is a peculiar form of truncal VM which must be distinguished from others that can show various combinations of the signs due to the aleatory mistaken embryogenic process..

So KTS is an anatomic and hemodynamic configuration that consist of combined a deep venous obstacle ( resistances to the draining flow responsible for osteo-hypertrophy) more or less perfectly compensated by superficial veins ( Open vicarious Shunt) as some times the marginal vein. The deep obstacles ( aplasia, hypoplasia) are more or less combined with venous incompetence. The superficial veins as the marginal vein are usually varicose and incompetent. Their hemodynamic “good” function ( Open Vicarrious shunt) and “bad” function ( closed shunts responsible for “Trendelenburg private circulation” varies according to the deep veins they connect. So, the “good” varicose veins should be preserved whilst the “bad” could be disconnected. Disconnected better than ablated because they drain the superficial capillary angioma which flow is relatively high ( residual pressure) and responsible for angioma color increase and varicose recurrence. For those reasons, an accurate diagnosis is mandatory, particularly differentiating the open vicarious shunts from the closed shunts, in order to elaborate the best strategy. DUS can help for such diagnosis thanks to provocative tests combined with the Perthes Manoeuver and plethysmographic changes at the calf when tiptoe elevating (or walking) under tourniquet placed just below the escape point of the shunts.

Claude

Dear colleagues,

Allow me a small excursion into the history.

Although Trendelenburg test assessing venous reflux and Perthes test assessing patency of deep veins constituted invaluable components of the functional examination of varicose veins before the era of DUS, both tests, unfortunately, were not generally used for the preoperative diagnostics. In many surgical departments, varicose veins were examined only by inspection, and pre-operatively marked on the skin; in addition, phlebography was performed to prove the patency of deep veins; venous reflux was often an unknown term. The standard varicose vein operation embodied ligation of the GSV in the groin, stripping, and excision of visible varices. Incompetence of the SSV remained mostly unrecognized; it was often neglected to perform flush ligation at the SFJ. As usual, varicose vein operations were carried out by surgical trainees. In the era of the boisterous development of vascular surgery, vascular surgeons scorned performing such banal operations. Prof. Bergan once pilloried the situation as follows: “It was regarded as impolite to speak about varicose veins on meetings of vascular surgeons”. It is therefore not surprising that the results of varicose vein operations were unsatisfactory.

Perthes, a German surgeon and disciple of Trendelenburg (who was chief of the Department of Surgery in Bonn, Germany) published his test in 1895 (Perthes G. Results of varicose vein operations according to Trendelenburg. Dtsch Med Wschr 1895;21:253-7 [German] ); the test offered the possibility to assess patency of deep veins before the era of phlebography. In the article, Perthes presented results of the Trendelenburg operation, which consisted of ligation of incompetent GSV in the lower half of the thigh. The disease was called that time varices cruris – varicose veins of the lower leg; thus, ligation of GSV above the knee was considered to be adequate; therapeutic results presented by Perthes – quick healing of varicose ulcers – justified the GSV ligation (many patients operated by Trendelenburg had varicose ulcers).

Trendelenburg himself described another – largely unknown – maneuver assessing patency of deep veins: the so called reversed Trendelenburg test: a tourniquet is placed onto the thigh in a standing position; the patient lies then down, and the limb is elevated; bulging varicose veins empty, although not so quickly as through the GSV, Trendelenburg stated. In this way, drainage through calf perforators into the deep veins and patency of deep veins could be demonstrated. Aside: could this test compressing the marginal vein with a digit be helpful in patients with CVM? Trendelenburg insisted on performing this test before each GSV ligation. At that time, i.e. in the 2nd half of the 19th century, surgery consisted usually of excision of varicose veins using long incisions, and leaving the stem of the GSV intact. It was assumed that ligation of GSV, i.e. of the main channel draining venous blood from varicose veins and from subcutaneous tissue of the lower extremity would aggravate venous congestion. Trendelenburg was the first who found that the flow in GSV in varicose vein patients was oriented predominantly in the retrograde direction, and that ligation of the incompetent GSV would repair the hemodynamic disorder. He knew that the retrograde flow in the GSV continued to stream further through calf perforators into deep lower leg veins; he named that pathological streaming, which we now call saphenous reflux, private circulation.

Regards,

C. Recek

**Dear colleagues,**

**Paradoxically, the Cestmir Closed Shunts denial re-enforces the Closed Shunt concept. Find below my reply.**

Cestmir Recek:I would like to outline my attitude toward the contentious topics.

Saphenous reflux, i.e. centrifugal streaming in incompetent GSV or recurrent reflux in GSV remnant after high ligation/crossectomy causes ambulatory venous hypertension; there is no doubt about it. This was documented by direct venous pressure measurements as well as by plethysmography registering hemodynamic disturbance – the correlate with ambulatory venous hypertension.

**Claude Franceschi: Nevertheless, the Perthes manoeuvre empties the previously dilated incompetent GSV ( which means less flow/less pressure) as well as the direct venous measurement shows a pressure drop at the ankle ( your attached figure)**

Cestmir Recek :As to competent/incompetent calf perforators:

There is a lot of communicating channels between deep and superficial veins in the lower leg. Nobody has as yet provided conclusive proof that these communicating veins as a whole enable only a unidirectional inward streaming from superficial into deep veins. This view formulated by Cockett has been based only on speculation, without being supported by any evidence; notwithstanding, it was generally accepted as a canonized article of belief. How incomprehensible soever it may appear, this is no exceptional happening, no rarity; in the history of medicine, mind-boggling, incredible rubbish was held for centuries as the official opinion/theory.

**Claude Franceschi:Nevertheless this incredible rubbish is proved every day by DUS , unless DUS is rubbish and not reliable. I suggest you to visit an hospital equipped with DUS used by an expert and note the exceptional refluxing perforators at the ankle in healthy people , except at the foot, the sole perforators are physiologically incompetent and refluxing.**

Cestmir Recek On the other hand, simultaneous pressure recordings in healthy people proved that pressure curves in PTV and GSV were nearly identical documenting free pressure and flow transmission in both directions, i.e. displaying pressure behaviour typical of conjoined vessels. This evidences that communicating veins are either valveless or have incompetent valves. By chance, one exceptional situation occurred (whatever the reason for it might be) that showed how the pressure curves in PTV and GSV would have looked like if the communicating veins had been fitted with competent valves hindering pressure transmission into the GSV: steep increase in pressure in the PTV but no increase in pressure in the GSV. This is the pressure pattern typical of the competent mitral valve in the heart: steep increase in pressure in the left ventricle, no increase in pressure in the left atrium.

I cannot imagine a more conclusive proof.

**Claude Franceschi: This is an opinion, not a proof.**

Cestmir Recek :I am afraid I must state that closed shunts are a fiction; they do not exist in reality. Existing pressure conditions don’t allow centripetal streaming in the popliteal and superficial femoral vein during calf muscle diastole. Ambulatory pressure gradient arising during calf muscle diastole entails higher pressure in the popliteal/femoral vein and lower pressure in deep lower leg veins; thus, it is impossible that centripetal flow could take place in the popliteal/superficial femoral vein during diastole to feed the centrifugal (refluxing) flow in incompetent GSV; this is verifiable by DUS.

**Claude Franceschi: Nevertheless, closed shunts exist. I sent in my previous mails a DUS video that “proved it” at the SPJ. Try it with BFLOW, in order to make sure you detect all velocities, low velocities particularly.**

Cestmir Recek :On the contrary, at the beginning of the diastole physiological**centrifugal** flow lasting 200-300 milliseconds occurs in the superficial femoral vein until the competent valves close (this flow is evoked as well by the ambulatory pressure gradient). Simultaneously with the physiological ***centrifugal*** flow in the superficial femoral vein, centrifugal refluxing flow fed from the iliac veins takes place in the incompetent GSV. After the centrifugal flow in superficial femoral vein has been stopped, the blood does not move here until the ambulatory pressure gradient is equalized or a new calf muscle contraction produces increase in pressure and propels the blood centripetally.

**Claude Franceschi: Nevertheless, this doesn’t contradict the Closed Shunt concept. It supports it**

Cestmir Recek :Assessment of functional venous volume is based on filling of veins in the lower limb from above: when a varicose vein patient lying with elevated limb stands up, emptied incompetent veins are filled from above; the same happens when performing Trendelenburg test. Furthermore, Trendelenburg documented in the late 19th century the filling and emptying of varicose veins by lowering and lifting the limb in a recumbent patient.

**Claude Franceschi: That is why CHIVA disconnects at the same time the Closed Shunts and divides the gravitational hydrostatic pressure ( so restoring the Dynamic Fractionation of the Gravitational pressure) by the mean of SFJ disconnection in case of GSV Closed shunt**.

Cestmir Recek **:Hemodynamically significant** **reflux** in the incompetent GSV with incompetent SFJ is fed from **incompetent** iliac veins (a condition typical of varicose vein disease); in incompetent iliac veins/inferior cava vein, sufficient amount of blood is available for forceful GSV reflux to counteract the ambulatory pressure gradient and to equalize the decreased pressure in lower leg veins; any competent valve situated above the SFJ (in common femoral, iliac vein) reduces the blood volume necessary to counterbalance the decreased pressure in lower leg veins. Blood volume contained between the competent valve in the common femoral/ iliac vein and the SFJ is too small to equalize the pressure gradient, and is therefore hemodynamically not fully effective.

**Nevertheless daily DUS shows a smaller volume of SFJ reflux when fed only by the deep veins below the this valve, BUT it exists, and confirms the Closed Shunt. If in this latter case the SFJ is not hemodynamically significant, why do the high Perthes ligation reduces dramatically the incompetent GSV calibre?**

**The Cestmir Closed Shunts denial paradoxically re-enforces the Closed Shunt concept as I define it, i.e:**

Closed Shunt : CS.

During the relaxation ( diastole) following the contraction (systole  that of the calf pump , the pressure at the entry of the calf pump is lower than at its exit  ( inverted Pressure gradient)  so triggering a back flow (diastolic reflux) immediately blocked by the valves closure in normal. In case of superficial valve incompetence of the superficial veins (N2 or N3)  and their connections ( Escapes points: perforators, pelvic leaks, SFJ,SPJ) ,  such closure is not achieved and part of the deep venous blood refluxes from the deep veins (N1) down to the calf pump entry through the escape point, then the incompetent superficial veins and finally the re-entry perforators. Part of the diastolic refluxing deep blood through the incompetent superficial veins is propelled upward during the systole and returns back again at the calf pump entry during the following diastole. This recirculation of deep blood was suspected by Trendelenburg ( Private circulation) but not proved because of lack of assessing means,  fortunately today proved by DUS. This recirculation is named Closed Shunt, because part of the deep blood is diverted by the superficial shunting veins (In fluid dynamics, Shunt = path diverting a flow)  in a closed loop ( Closed Shunt). The amount of shunted deep venous blood, varies according to the calf pump efficacy and the resistance of the shunting superficial veins, escape point and re-entries. It depends also on the deep blood volume available during the diastole. E.g, when the escape point is the SFJ,  the refluxing deep blood volume will be limited to the blood below the SFJ when the overlying femoro-iliac valve is competent and overloaded by part of the ilio-femoral flow when the femoro-iliac valve is incompetent.

**May be some disagreements are due to Babel’s misunderstanding , particularly when at least one of the partners ( me) doesn’t speak/understand properly the English.**

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**Open Deviated Shunt:ODS**

An ODS is made , for example, of a GSV incompetent tributary refluxing down to a below knee perforator re-entry in absence of SFJ or any other escape point reflux ( e.g a competent terminal valve). In that case, the varicose tributary is overloaded during the calf diastole , only by the proper GSV skin draining flow and not by any deep venous flow. So, there is no recirculation of deep nor superficial blood. The reflux is just due to the tributary incompetence that allows a reflux instead of a normal drainage through the SFJ, because the pressure in the calf pump is lower than inside the femoral vein (competitive pressure gradient). So the incompetent tributary plays the role of a shunt that “deviates” the normal draining flow down to the re-entry instead of up to the SFJ. This shunt is “open” because there is NO recirculation . This peculiar and frequent ODS is called Shunt II in the CHIVA shunts classification. Its disconnection, restores a normal GSV flow direction and limits the remaining tributary refluxing flow to the physiologic drainage of its skin territory, so reducing to normal its caliber.

**Open Vicarious Shunt:**

In case of venous obstruction, the skin draining flow forces a compensatory “vicarious” collateral , so called “vicarious “ shunt that is open because it doesn’t cause any “recirculation”. It occurs in case of pathological obstruction, but also when it is due to superficial veins ablation , responsible for part of recurrence. Such recurrence by OVS, is prevented by the respect of the varicose veins.

I apologize for this “jargon”…but new terms are necessary to name new or upgraded concepts. Greeks called “barbarian” people who spoke a foreign language they didn’t understand. English is every day less barbarian to me in proportion to my improvement in understanding it.

Regards

Claude