PRESIDENTIAL ADDRESS

The dynamic approach to venous disease—following in the footsteps of Gunnar Bauer and Robert Kistner

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It is a privilege for me to address you during this Seventeenth Annual Meeting of the American Venous Forum (AVF). It was a great moment of my life when you chose me as your president-elect in Cancun, Mexico, in 2003, and I was escorted to the podium by Anthony Comerota and Robert Kistner. The last year has meant intense work with our excellent board, particularly monthly conference calls across the Atlantic with the executive committee, including Thomas Wakefield, Frank Padberg, Joann Lohr, Joseph Caprini, and Robin Hoyle. Last September at the retreat with the board in Washington, DC, the potential new directions for AVF were discussed:

- develop a new format for the annual meeting to accommodate the educational needs of vascular surgeons in training,
- work with the program directors in writing a venous curriculum as part of the certificate in vascular training,
- establish a venous screening program with the American Vascular Association, and
- build partnerships with other venous societies such as the American College of Phlebology, the European Venous Forum, and the Venous Forum of the Royal Society of Medicine.

I am living in southern Sweden on the shore of Öresund, the busy waterway between Denmark and Sweden. To the right, I see Helsingör in Denmark with Hamlet's castle, Kronborg, and to the left, in the middle of the sound, the island of Ven. Ven is Swedish for *vein*, so every morning when I open my eyes, I am reminded of veins.

The island of Ven is famous for being the home of Tycho Brahe (1546-1601), the Danish nobleman and astronomer who was fighting other stargazers like Copernicus, Kepler, and Galilei about basic laws of the universe, such as whether the sun was circling the earth or vice versa. Sir Isaac Newton (1642-1727), the English father of modern science, was referring to Tycho Brahe and his colleagues in his famous expression "If I have seen further (than others), it is by standing on the shoulders of giants."

Stimulated by the island of Ven, I would like to present two pioneers whose groundbreaking work is the foundation of modern phlebology and an inspiration for all of us involved in the management of venous disease—Gunnar Bauer and Robert Kistner—proving my point of "one giant standing on the shoulder of another."

GUNNAR BAUER (1895-1970)

Gunnar Bauer, the father of Swedish phlebology, attended medical school at the University of Lund and Karolinska Institute in Stockholm and graduated in 1922. He started his surgical career with Clarence Craaford, the Swedish pioneer in cardiothoracic surgery at Sabbatsberg Hospital in Stockholm. During 1926-1933, he continued his surgical training at the hospital in Malmö, where he also defended his >1,000-page PhD thesis on peritonitis after appendicitis. After a few years in Sveg, a tiny hospital in northern Sweden, he moved to Mariestad in southern Sweden in 1937, where he stayed until his retirement in 1960. He was the only physician at this small hospital for many years.

The lack of assistance and inspiration from other colleagues, in addition to their criticism, makes his contributions even more impressive. Despite working at this remote hospital, his collaboration with Craaford's group in Stockholm continued. Bauer had an early interest in venous thromboembolism. Two medical milestone events paved the way for his future: the unique contributions of Jorpes' purification of heparin in 1935 and Dos Santos' development of ascending venography in 1938.

Bauer's contributions include his venographic description of

- the normal venous system of the leg,
- acute thrombosis of the leg, post-thrombotic (secondary) deep venous incompetence, and

From the University of Lund.
Competition of interest: none.
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- idiopathic (primary) deep venous incompetence.

His treatment contributions include

- treatment of acute thromboembolism with heparin, and
- treatment of idiopathic (primary) and post-thrombotic (secondary) deep venous incompetence with resection of the popliteal vein.

The normal venous system. The same year as Dos Santos published his paper, Bauer perfected the technique of venography and started to use it in his clinical practice. The venographic image of the normal venous system was practically unknown, but Bauer outlined the typical duplicated veins following the three arteries in the calf with their densely arranged valves, and the solitary popliteal and femoral veins with fewer valves.

Acute thrombosis. Before Bauer's publications it was commonly supposed that the thrombotic process started in the femoral or pelvic veins and then extended retrogradely into the deep leg veins. Bauer was able to demonstrate that this was not true and that:

- a thrombosis in the calf veins is more common than previously believed,
- the thrombotic process starts in the calf veins in most cases,
- the acute thrombosis can be accurately diagnosed in its earliest stage by venography, and
- the evolving thrombotic process and the final post-thrombotic state can be studied by venography.

When Bauer's first publication appeared in 1940[1] it described discoveries of the utmost importance, not only for scientific enlightenment but also for clinical practice. Bauer particularly emphasized the importance of detecting the early, easily overlooked clinical signs and how these signs corresponded to the venographic findings. In a 10-year follow-up of 100 patients with deep vein thrombosis (DVT) who did not receive any treatment,[2] he found that all had swelling of the leg, 90% had skin induration, and 80% had venous ulcers with severe pain and a risk of fatal infections. He found that if the valves in the popliteal vein were preserved, the post-thrombotic syndrome developed in very few patients, and he envisioned:

> Is it not possible to intervene in the course of developments and prevent the popliteal vein from being invaded by the thrombus? With venography the thrombus can be diagnosed while it is still localized to the calf veins. Why cannot some means be developed by which to prevent its further spread upwards? If this could be done, it would also mean that the late results of thrombosis could be radically changed. A prophylactic against the post-thrombotic disease would have been found.

The post-thrombotic (secondary) deep venous incompetence after acute DVT. Already in 1866, John Gay had pointed out that skin changes and leg ulcers were, in his experience, invariably associated with post-thrombotic disease of the deep veins. This concept was reiterated by Homans, who believed that post-thrombotic damage to the deep veins was the probable cause of leg ulcers and that so-called venous stasis was the crucial factor. Before 1940, the sequelae of acute thrombosis were relatively unknown. Chronic edema, eczema, and ulceration were thought to be caused by varicose vein disease.

Clinical and venographic studies convinced Bauer that the post-thrombotic syndrome developed because the initially effective subcutaneous venous collaterals dilate and become varicose as a result of overloading. He changed his initial belief that the deep veins remained occluded after DVT because of findings obtained with a new technique: descending transfemoral venography in the upright position, which showed that recanalization of the deep veins was the rule and permanent occlusion the exception, and also that the valves were destroyed during this process. Bauer appears to have been the first to state that the destruction of the deep vein valves was the reason for the development of post-thrombotic disease. He was convinced that the deleterious change was the conversion of the valve-bearing popliteal and femoral veins into a valveless tube, resulting in a dysfunctional calf muscle pump.

Idiopathic (primary) deep venous incompetence. Bauer agreed with Homans that in almost every case of severe chronic venous insufficiency with pain, swelling and ulceration, the cause was "venous stasis" resulting from valvular incompetence of the femoral vein. On descending venography in 55 patients with deep reflux,[3] he surprisingly noted that in 30 limbs it was "not caused by thrombosis, a condition which seems to be very little known," both then, in1948 as well as now, in 2005. In the absence of the exact knowledge of the etiology, he termed this condition *idiopathic*:

- no history of DVT;
- hereditary disposition for varicose veins;
- female predominance, but not related to pregnancies;
- clinical picture the same as in post-thrombotic patients;
- venographic picture markedly different from post-thrombotic patients;
- the femoral vein was generous with uniform width;
- abundant valve formations seen;
- contrast often followed down to the ankle;
- macroscopically normal-looking vein;
- microscopically thickening of the intima of the venous wall with hyaline fibrosis; and
- marked fibrosis in the media, with splitting of the elastic elements and atrophy of the muscular layer.

Bauer's interpretation was that there was a loss of elasticity in the vein wall leading to dilatation and subsequent incompetence of the valve. A vicious circle is started with reflux leading to further dilatation. He regretted his own lack of knowledge about the microscopic state of the valves.

Treatment of acute thrombosis with heparin. Heparin was discovered in 1916 by Jay McLean, a second-

year medical student at Johns Hopkins University. Eric Jorpes at Karolinska Institute in Stockholm defined the chemical structure and purified heparin in 1935. Simultaneously, Jorpes and Craaford in Stockholm and Murray and Best in Toronto tested heparin in thromboembolic disease. Based on a large controlled study in Stockholm on 627 patients operated on between 1937 and 1940, Craaford[4] showed that prophylactically given intravenous heparin starting immediately after surgery with a 7-day duration totally eliminated thromboembolic complications and mortality after major surgery. Aware of this study, Bauer in Mariestad, who had started to use venography to diagnose DVT, now introduced the epoch-making treatment of venous thromboembolism (VTE) with heparin.

Jorpes, in his 1946 book on heparin, acknowledged Bauer's role in the treatment of VTE: "The most valuable contribution to the development of this question has been made by Gunnar Bauer."[5] Michael DeBakey from Houston confirmed Bauer's role in a personal conversation in 2003 in Lund, Sweden.

Bauer's principles were early diagnosis confirmed by venography, intravenous heparin treatment, early mobilization, and compression bandaging. At a meeting in Stockholm on December 1, 1940, at the same time as Craaford presented his findings of prophylaxis, Bauer reported his early results on 21 patients with calf vein thrombosis treated with this model compared with 32 patients with calf vein thrombosis before he had access to heparin (Table)[6] In the heparin treated group, none had proximal extension compared with 75% in the nontreated group; none had clinical pulmonary embolism compared with 34%; none developed contralateral DVT compared with 31%; and the bed rest was 7 days compared with 43 days.

These results were sensationally good, and Bauer explained that was due to the ability to detect the thrombosis in its earliest state by venography, thus allowing for the administration of heparin earlier than was previously possible. Bauer continued to treat his patients according to his principle, and in 1964, he published 937 cases of thrombosis with only seven fatal pulmonary embolisms (<1%).[7]

Treatment of the idiopathic (primary) and post-thrombotic (secondary) deep venous incompetence with resection of the popliteal vein. Having identified deep reflux as the main culprit in severe chronic venous insufficiency—be it of primary or secondary etiology and believing in the crucial role of popliteal vein reflux—Bauer took the next step in treatment and recommended resection of the popliteal vein to stop the reflux into the calf veins.[3] Homans and Linton in Boston had advocated femoral vein ligation for similar reasons. However, Bauer argued that the ligation of the femoral vein distal to the confluence of the profunda was inadequate because it allowed reflux through the profunda vein to communicate with the distal femoral vein, creating another valveless axial reflux stem to the calf.

Popliteal vein resection turned out to have low morbidity. The patients wore a compression bandage for at least 2 months, sometimes much longer. Bauer reported marked improvement in all patients. The aching, bursting pain was relieved at once, and the ulcers generally healed in a shorter time than after conservative treatment. He published >500 cases and performed >1,000 popliteal vein resections in Mariestad. His credo, though, was that the best way to control the post-thrombotic syndrome was to prevent its development by treating the acute thrombosis immediately and aggressively, hoping to prevent the development of a valveless femoral tube.

Early results of Bauer's study in patients with calf vein thrombosis treated with heparin*

	No heparin (%)	*Heparin*
No. of cases	32	21
No. with proximal extension	24 (75)	0
Clinical PE	11 (34)	0
No. of cases with contralateral leg DVT	10 (31)	0
Mortality VTE	2 (6)	0
Bedrest, days	43	7

PE, Pulmonary embolism; *DVT*, deep vein thrombosis; *VTE*, venous thromboembolism.
*Table modified from ref 6.

Gunnar Bauer criticized. Naturally, the Swedish radiologists criticized this Swedish surgeon (not an unusual phenomenon) for his venographic technique with the patient supine, where he may have misinterpreted filling defects as thrombus and consequently obtained excellent results with heparin therapy. Many also maintained that the excellent initial results of popliteal vein resection in patients with severe chronic venous insufficiency could be related to the efficient postoperative compression treatment and careful wound care. Thirty-eight of Bauer's original patients were followed up 20 years later by Hallböök and Lindhagen.[8] All but two patients had a relapse of their previous problems, and >50% had recurrent venous ulcers. Repeat venography showed that refluxing collaterals had developed, bypassing the resected popliteal vein. In a commentary, John Bergan wrote ". . .the last outpost of vascular ligation finally surrendering."

Gunnar Bauer, the Swedish pioneer. Gunnar Bauer, the first modern phlebologist, introduced proper diagnosis and directed treatment for acute and chronic venous disease. He described the venographic findings of the normal and thrombosed veins in the leg and how fresh thrombi progressed from the calf veins into the proximal leg veins. He elucidated the recanalization process of the occluded deep veins and proved the connection between the post-thrombotic reflux due to destroyed venous valves and chronic venous insufficiency. He was the first to describe idiopathic (primary) deep venous incompetence, a shoulder for Robert Kistner to stand on, introducing venous valve reconstructions. Bauer revolutionized the management of patients with acute DVT by introducing venography for early diagnosis and intravenous heparin combined with early mobilization and the compression bandage for treatment.

What he taught us about pathology, diagnosis, and treatment marks a turning point in phlebology. Gunnar Bauer's contribution was significant, and he worked hard as a missionary to promote his gospel: he published 100 papers in the phlebologic field and gave numerous lectures in Sweden as well as abroad. With Knut Haeger, he created Societas Phlebologica Scandinavica in 1963, where until his death in 1970, he continued to inspire research and education in the true spirit of the American Venous Forum.

Through his son, Hans Bauer, I got a copy of all of Gunnar Bauer's publications, which I brought with me to Kuwait with the idea to write a biography of his pioneering works. Unfortunately, I lost all of my documents during the occupation of Kuwait by Saddam Hussein in 1990. The Iraqi dictator apparently had a special interest in venous disease.

ROBERT KISTNER

Standing on the shoulders of Gunnar Bauer is another giant, Robert Kistner, the pioneer for venous reconstructive surgery, who got his medical degree from the medical school at St Louis University in 1954. After surgical residency in St Louis, he passed his surgical board in 1961 and completed his vascular surgery training with a fellowship at the Cleveland Clinic in 1965. Since 1966, Robert Kistner has been working at Straub Clinic and Hospital in Honolulu, Hawaii, where all his pioneering work on venous disease has taken place:

- internal valve repair,[9]
- descending venography to classify the severity of reflux,[10]
- transposition of the incompetent deep vein into a valve bearing segment,[11]
- external valve repair,[12] and
- endophlebectomy.[13]

I have had the privilege to know Robert Kistner since 1982, when he was the first visiting professor during my tenure as professor and chair of surgery at the University of Kuwait (1981-1990). When Saddam Hussein ended my Middle East career in 1990, Bob offered me an opportunity to join him in Hawaii, where I started at Straub Clinic and Hospital in February 1991, which gave me more than 12 years of collaboration in this venous Mecca.

Internal valve repair. At the postgraduate course for the AVF in February 2004, Kistner gave a presentation on "From Serendipity to Practicality: Fundamental Questions Generated by the First Successful Valve Repair in 1968." He described his first patient, who suffered swelling and pain of the extremity within 30 minutes of getting out of bed and was disabled from work. He had apparently had DVT 2 years before. An ascending venogram showed patency of the deep veins, incompetent perforator veins of the calf, and evidence of a recanalized femoral vein. A descending venogram showed severe reflux in the femoral vein extending into the crural veins, with no obstructive elements. Surgical repair of the proximal femoral valve was performed 4 days after calf perforator interruptions and was followed by immediate relief of pain and swelling and subsequent return to work. A postoperative venography proved the valve competent and the deep veins patent. The patient was followed for 12 years, without recurrence of symptoms. Kistner wrote:

> Were it not for a number of fortunate events that combined to make the first case successful, it is likely that the subsequent vigorous attempts to do more cases would have been blunted or discarded. The case began with the good fortune that this man had a repairable valve high in the femoral vein combined with more distal deep vein thrombosis. The valve was entirely normal except for a floppy cusp when the vein was opened (subsequently termed primary valve disease). There were no visible signs that the venous thrombosis had extended to the level of this valve. The distal femoral vein and the popliteal vein were patent, but were interpreted as being post-thrombotic. A whole series of additional fortunate events occurred as the diagnostic workup and the newly devised surgical repair were carried out, and the ultimate fortunate event was that the procedure was uncomplicated and dramatically successful in relieving the patient's disability.

Kistner's 1968 report[9] of the first valve repair came 20 years after Bauer's description of idiopathic valvular incompetency. However, the control of reflux by Bauer's destructive resection of the popliteal vein had now been replaced by Kistner's reconstructive repair of the floppy valve. After the success of this first case, Kistner realized the need for an accurate workup of patients with severe chronic venous disorders (CVD) to separate reflux problems from obstructive problems, to localize the sites of potentially repairable reflux, and to identify whether the reflux was due to "primary" valve cusp stretching or to post-thrombotic scarring and destruction of the cusp.

Descending venography to classify the severity of reflux. Bauer had developed the technique of descending venography in the standing position. He had shown the difference between post-thrombotic deep venous reflux with valve destruction and idiopathic valve incompetence with uniformly widened femoral vein, abundant valve formations, and reflux, often to the ankle. This technique was further refined by Kistner, who developed the Straub technique,[10] where descending venography is performed on a fluoroscopic table capable of a 60° tilt. The procedure is videotaped to provide a permanent record and for later review.

A catheter is percutaneously introduced from the contralateral common femoral vein and passed across the inferior vena cava confluence to study the affected extremity. This allows selective catheterization of the major veins of interest. The reflux is classified into 4 grades, where grade 1 is a wisp of reflux and grade 4 is cascading reflux into calf.

The purpose of descending venography is to establish the ability of proximal valves to close and prevent reflux. If no valves are competent, each of the refluxing valves is clearly seen and the operability of the valve is established.

This investigation also differentiates between primary and secondary valvular incompetence.

Reconstruction for deep venous reflux. In Kistner's long-term results (4 to 21 years of follow-up),[14] three groups could be distinguished: primary valvular incompetence (PVI) with >70% good-to-excellent clinical outcome; secondary (post-thrombotic) valvular incompetence (SVI), where mainly transposition was performed with <40% positive outcome; and finally, the combination PVI/SVI where >60% good-to-excellent clinical outcomes were achieved.

Kistner stimulated many followers who have reported their own series: DePalma, United States; Eriksson, Sweden; Gloviczki, United States; Hoshino, Japan; Lurie, Russia; O'Donnell, United States; Perrin, France; Raju, United States; Sottiurai, United States; Taheri, United States; and Tripathi, India. In 2004, Perrin[15] published an excellent review on venous reconstruction for deep venous reflux.

Reconstruction for deep venous obstruction. The combination of proximal, mainly iliac vein obstruction and distal deep venous reflux leads to the highest degree of venous hypertension and most severe postthrombotic syndrome. The original bypass procedures— femorofemoral for iliac vein occlusion and saphenopopliteal for femoral vein occlusion—are today almost obsolete. Iliocaval obstructions are currently treated by angioplasty and stenting. Raju and Neglén[16] have performed >700 procedures with a cumulative secondary patency rate of 90%. Kistner's group[13] has shown that surgical endophlebectomy of post-thrombotic veins is feasible as an adjunct to deep venous reconstruction, particularly to improve the inflow before iliac vein angioplasty.

The surgical release of synechiae, septae, and endoluminal masses significantly increases diameter, compliance, and flow in treated vein segments as observed by duplex scans. As Bauer had already stated, the best way to avoid iliac vein obstruction is aggressive treatment of the acute thrombotic episode with early thrombus removal, which today is possible by catheter-directed thrombolysis, percutaneous thrombectomy, or open thrombectomy.

Robert Kistner, the American pioneer. Kistner, like Bauer, based his development in the management of venous disease on proper diagnosis followed by precise treatment. At the Straub Clinic and Hospital in Honolulu, Hawaii he created a true venous center for the management of acute and chronic venous disease, where the Straub Foundation was the base for research and education. It has had a venous research fellowship program since 1990, with the following fellows: Elna Masuda, Howard Wong, Dean Sato, and Nahidh Hassaniya, United States; Danian Yang, China; Berndt Arfvidsson, Sweden; Gudmundur Danielsson, Iceland; Tomohiro Ogawa, Japan; Alessandra Puggioni, Italy; and Bert van Gent, The Netherlands.

After the introduction of the three reconstructive procedures—internal and external repair of primary valvular incompetency and transposition in secondary valvular incompetency—the need for improved diagnostic tools was evident. There was no standardization of the noninvasive tests in different vascular laboratories and no accurate classification, so that badly needed interinstitutional trials were difficult to conduct. Kistner realized this need, and he was one of the initiators of the 1994 CEAP consensus meeting on Maui under the AVF umbrella. Kistner is currently active with Fedor Lurie in evaluating the function of the venous valve in health and disease.[17-20] Like Bauer, Kistner is a missionary travelling the world, promoting his gospel that "specific diagnosis and specific treatment in CVD is a concept whose time has now come."[21]

Aggressive treatment of iliofemoral venous thrombosis. Others have followed in the footsteps of Gunnar Bauer and Robert Kistner. Bauer established the importance of early diagnosis of DVT using venography, instituting heparin treatment, and early ambulation to avoid the post-thrombotic syndrome. Kistner demonstrated that the results after venous reconstructive surgery were superior in patients with primary deep valvular incompetence compared with patients with secondary (post-thrombotic) incompetence. It therefore seems logical to assume that the initial removal of the thrombus should be most advantageous.

The importance of early thrombus removal has recently been further emphasized by studies on the inflammatory response to DVT. Wakefield and his group[22] in Ann Arbor have shown that the leucocyte adhesion molecule P-selectin activates the leucocytes migrating into the venous wall, creating an inflammation that destroys the venous wall and the valves. Harris and his group[23] from Stanford showed in another experimental model that if the thrombus was removed, the inflammatory changes were reversible. The importance of the proximal obstruction for the development of distal valvular incompetence has been carefully studied by Strandness and his group in Seattle. In a series of articles, they have shown that

- from 20% to 50% of initially uninvolved distal veins became incompetent by 2 years[24];
- the combination of reflux and obstruction, as opposed to either alone, correlated with the severity of symptoms and was present in 55% of symptomatic patients[25];
- 25% of all venous segments developed reflux in time, of which 32% were documented to not have been previously involved with thrombosis[26]; and, finally
- in a study of the posterior tibial veins located below a popliteal segment involved with thrombosis, 55% of the distal veins became incompetent if the segment remained obstructed, compared with 7.5% of those below a popliteal vein that recanalized.[27]

These changes in the distal veins occur early enough that they can't be blamed on proximal valvular reflux, although this also comes into play with time. Thus, it would appear that the early relief of obstructing thrombus by thrombolysis or thrombectomy should prevent more extensive post-thrombotic sequelae, if only by protecting the distal veins against progressive valvular incompetence. This is a pivotal point that has not been well recognized, not

only in terms of basic pathogenesis but also in judging the results of thrombolysis and thrombectomy, where critics have largely ignored the restoration of proximal patency while focusing on the presence or absence of valve reflux. The conclusions from the studies cited above are clear: early removal of the proximal thrombus conveys significant benefits, and the earlier the removal, the better the outcome.

In this address, I will focus on the development of venous thrombectomy, which is a valid alternative if catheter-directed thrombolysis or percutaneous mechanical thrombectomy fail or are contraindicated.

The first thrombectomy for iliofemoral DVT was performed by Läwen[28] in Germany in 1937. In 1940, Homans discussed indications for thrombectomy with heparinization, with or without ligation of the femoral vein.[29] The modern era for thrombectomy in the United States started with Mahorner's 1954 article "New Management for Thrombosis of Deep Veins of Extremities"[30] in which he advocated for thrombectomy and regional heparinization and presented five of six patients with excellent clinical results. The enthusiastic wave created by the Mahorner blues, and especially boosted by the Haller and Abrams[31] reports in 1963, was efficiently quelled by Lansing and Davies[32] in 1968 with their 5-year follow-up of the Haller and Abrams patients.

One of the most enthusiastic groups came from Strong Memorial Hospital in Rochester, NY. I had the privilege to spend a year as research fellow there in 1968 to 1969. It was an excellent academic vascular center under the leadership of Charles Rob. For many years, it was also a center for venous disease, with pioneers such as James Adams, Andrew Dale, James DeWeese, Walter Pories, and Seymour Schwartz.[33]

I returned to the University of Lund, Sweden ready to perform venous thrombectomies. Inspired by a report from Jean Kunlin[34] on the use of a temporary arteriovenous (TAV) fistula to improve outcomes of venous reconstructive surgery and the application by Jörg Vollmar and Henning Loeprecht of this technique, the first iliofemoral venous thrombectomy with a TAV fistula was performed in 1974. My personal experience of this surgical procedure now comprises >200 patients: 70 in Lund, Sweden (1974-1978); 31 in Helsingborg, Sweden (1978-1981); 102 in Kuwait (1981-1990); and 10 in Hawaii (1991-2003).

Four PhD theses were partly based on this development at the University of Lund.[35-38] We performed a prospective, randomized study comparing thrombectomy plus TAV fistula with conservative treatment in patients with acute iliofemoral DVT, with follow-up after 6 months, and 5 and 10 years.[39-41] We found a significantly better patency of the iliac vein in the surgical group, which also showed better competency of the deep veins.

Continuous improvement of the surgical technique has taken place. During my 9 years at the University of Kuwait, we were consulted on >400 patients with acute iliofemoral DVT, mainly young women during or after pregnancy. Together with the Indian coagulationist Dipika Mohanty, we started a prospective study on coagulation and fibrinolytic factors in women with normal pregnancies and DVT. We had protocols for >100 individuals in our computer system, which was taken to Iraq at the time of invasion 1990, another proof of Saddam Hussein's interest in venous disease.

The Czech interventional cardiologist Jiri Endrys, who worked in our team, developed a percutaneous technique to occlude the TAV fistula via the untouched contralateral femoral artery by placement of inflatable balloons into the fistula.[42] Simultaneously, venography was used to study the proximal thrombectomized iliac vein, and in case of obstruction, an angioplasty could be performed, which today would be combined with stenting.

Drs Al Hassan, Christenson, Nazzal, and Neglén studied the effect of extension of the iliac clot into the inferior vena cava (IVC) in 52 patients.[43] They showed that *all* patients with failed removal of the IVC clot developed DVT in the contralateral leg compared with one patient (7%) in the group with successful IVC thrombectomy.

The procedure of thrombectomy plus TAV fistula provides the vascular surgeon with an interesting combination of specific open surgical technique with modern catheter-based adjunctive techniques to improve the outcome:

- Fogarty balloon catheter;
- intraoperative venogram, angioscopy, or intravascular ultrasound scan to check for iliac vein compression or remaining thrombus;
- intraoperative angioplasty and stenting;
- intraoperative regional thrombolysis;
- insertion of a temporary IVC filter;
- arteriovenography to check TAV fistula and thrombectomized iliac vein postop; and
- percutaneous closure of TAV fistula.

The role of American Venous Forum to support the dynamic development of the venous disease. The Tower of Babel was created by the descendants of Noah to reach the sky, but the Lord punished the arrogant people by splitting their common language and spreading them from Babylon all over the world (Genesis 11:1-9). Saddam tried to rebuild the tower, which was supposed to be a large cube with each side 92 m long, and you all know how the wrath of God came down on him.

AVF has tried to tear down this Tower of Babel and create the CEAP classification as a common language for chronic venous disorders, which previously suffered from a lack of precision in description. This deficiency led to conflicting reports in studies of the management of CVD at a time when new modalities were being offered to improve treatment for simple as well as more complicated venous diseases. It was believed that these conflicts could be resolved by precise diagnosis and classification of the underlying venous problem.

Although there have been several classifications of CVD in the past—the most well known is the Widmer classification—none provided the completeness and objectivity needed for scientific accuracy. At the Fifth Annual Meeting of the AVF in Orlando, Fla, in 1993, John Porter

suggested using the same approach as TNM for cancer or CHAT for carotid artery disease in developing a classification system for CVD.

After a year of intense discussions, a consensus conference was held at the Sixth Annual Meeting of AVF on Maui, Hawaii, in February 1994, where an international ad hoc committee, chaired by Andrew Nicolaides with representatives from Australia, Europe, and the United States, developed the first CEAP consensus document.[44] The classification was based on clinical manifestations (C), etiologic factors (E), anatomic distribution of disease (A), and the underlying pathophysiologic findings (P), thus the name CEAP. I tried to get "history" in as one factor, but the committee did not like a *CHEAP* classification. The CEAP consensus statement was published in 26 journals and books and translated into nine languages (reflecting the Babel effect). Today, most articles about CVD use all or portions of CEAP.

Diagnosis and treatment of CVD is developing rapidly, and the need for an update was obvious when Peter Gloviczki in April 2002 appointed an ad hoc committee on CEAP to review the classification and make recommendations for change by 2004, 10 years after its introduction. An international ad hoc committee was also established to assure continued utilization. The committees held four meetings to revise the document, which was published in the *Journal of Vascular Surgery* in December 2004.[45] The recommended changes include:

- refinements of several definitions used in describing CEAP,
- refinement of the C-classes of CEAP,
- addition of the prescript "n" (no venous abnormality identified),
- incorporation of the date of classification and level of investigation, and
- description of basic CEAP introduced as a simpler alternative to the full (advanced) CEAP classification.

For the practicing physician, CEAP is an instrument for correct diagnosis to guide treatment and assess prognosis. For the researcher and reporting standards, CEAP aids to group the patients so that we are looking at the same type of patients. It is important to state that CEAP is a descriptive classification and a base for diagnosis and prognosis, whereas venous severity scoring and quality-of-life estimation are instruments for longitudinal research to assess outcomes. The new venous severity scoring system produced by the AVF ad hoc committee on outcomes under the leadership of Robert Rutherford[46] was intended to complement the descriptive CEAP classification.

It is up to the AVF and its outcomes committee to continue its important work to standardize the tests for CVD. Quoting Robert Kistner from his presidential address:

> Until the use of the CEAP classification for completeness of diagnosis and the employment of reliable tests to accurately identify pathologic problems are in place, we will not be able to conduct the large scale clinical studies with the statistical power afforded by larger numbers that will lead to sustainable conclusions about the relative values of various treatment methods in clinical practice.[21]

In December last year, I attended an excellent consensus meeting on Management of CVD of the Lower Limbs, organized by Andrew Nicolaides on Cyprus. I was disturbed by the repeated statement "the cornerstone for management of CVD is compression therapy." This is an archaic, counterproductive, and defensive opinion. With all the new armamentaria available to diagnose and treat CVD, I would state that the "cornerstone for management of CVD is an accurate diagnosis and classification of the underlying venous problem, which creates the base for correctly directed treatment." Compression therapy is certainly one aspect of the treatment, but is not the cornerstone anymore!

A spiritual reunion on the island of Ven. Robert and Adelaide Kistner came to Tycho Brahe's island of Ven in 1982 as the international guest speaker for Societas Phlebologica Scandinavica, the society that Gunnar Bauer established in 1963, thereby creating a spiritual reunion between the three pioneers and giants, Tycho Brahe, Gunnar Bauer, and Robert Kistner.

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