Thrombosis Research 182 (2019) 194–204

Contents lists available at ScienceDirect

Thrombosis Research

journal homepage: www.elsevier.com/locate/thromres

Review article

Venothromboembolic signs and medical eponyms: Part I

Fan Ye[a], Halil Tekiner[b], Eileen S. Yale[c,*], Joseph J. Mazza[d], Carolyn Stalvey[e], Steven H. Yale[f]

[a] University of Central Florida College of Medicine, Graduate Medical Education, 6850 Lake Nona Blvd, Orlando, FL 32827, United States of America
[b] Erciyes University School of Pharmacy, Department of the History of Pharmacy and Ethics, Kayseri 38280, Turkey
[c] Department of General Internal Medicine, University of Florida, College of Medicine, 2000 SW Archer Rd., Gainesville, FL 32608, United States of America
[d] Clinic Research Institute, 1000 North Oak Avenue, Marshfield, WI 54449, United States of America
[e] Department of General Internal Medicine, University of Florida, College of Medicine, 7540 W. University Ave., Gainesville, FL 32610, United States of America
[f] University of Central Florida College of Medicine, Department of Internal Medicine, 6850 Lake Nona Blvd, Orlando, FL 32827, United States of America

ARTICLE INFO

Keywords:
Venothromboembolism
Eponyms
History
Signs
Virchow

ABSTRACT

Eponyms are honorific terms ascribed to individuals who discovered a sign, test, syndrome, technique, or instrument. Despite some contentions, eponyms continue to be widely ingrained and incorporated into the medical literature and contemporary language.

Physical signs are considered unreliable methods alone for detecting deep venous thrombosis (DVT). The accuracy of the majority of these signs is unknown. For those signs that have been studied, there are a number of methodological limitations hindering the ability to draw meaningful conclusions about their accuracy and validity in clinical practice. Nevertheless, some findings when present and used in conjunction with other key signs, symptoms, and aspects of the patients history may be useful in further supporting the clinical suspicion and likelihood of DVT and/or pulmonary embolism (PE) or venothromboembolism (VTE). These signs also provide the means to better recognize the relationship between clinical findings and VTE. The acquisition of historical knowledge about these signs is important as it further enhances our understanding and appreciation of the diagnostic acumen that physicians were required to employ and to diagnose VTE prior to the advent of advanced imaging methods. Described in this paper is a brief overview of thrombosis as enumerated by Rudolf Virchow, and eponymous signs described in the late eighteenth and nineteenth centuries.

1. Introduction

Prior to the advent of advanced imaging techniques, physicians devised a variety of physical examination maneuvers described as signs of medical eponyms, as methods to detect lower extremity deep vein thrombosis (DVT) and pulmonary embolism (PE) or venothromboembolism (VTE). Eponyms are present in the medical literature because they reflect a simpler way of describing complex diseases, structures, or signs. They are honorific terms ascribed to individuals for their accomplishments, which may include identifying a sign, test, procedure, syndrome, medical device, or surgical technique.

Eponymous signs of venous thrombosis of the lower extremities were devised based on enumerating distinct clinical features found on physical examination including swelling, increased temperature, pulse, venous pain, and tenderness through palpation, compression, or movement. Symptoms when present are caused by functional obstruction and/or inflammatory changes within the vessel wall.

The paper first provides the readers with a historical perspective and framework of the three factors, as described by Rudolf Virchow, required for thrombosis formation, followed by presenting a brief biographical information, signs as originally described, and if available, there sensitivity and specificity. We present these signs chronologically based on the year they were first reported. Our goal is to recognize those pioneers whose aim was to advance the field of medicine by identifying methods to diagnose VTE, provide insights regarding the anatomical relationship and pathophysiological processes proposed for each sign, and determine the application of the sign in clinical practice.

2. Methods

PubMed, Medline, online internet word searches, and bibliographies from source text and textbooks were used. PubMed was searched using

Abbreviations: DVT, deep venous thrombosis; PE, pulmonary embolism; VTE, venothromboembolism

* Corresponding author at: University of Florida College of Medicine, Department of Internal Medicine, 2000 SW Archer Rd, FL 32608, United States of America
E-mail addresses: htekiner@erciyes.edu.tr (H. Tekiner), eileen.yale@medicine.ufl.edu (E.S. Yale), Mazza.joseph@marshfieldreseaerch.org (J.J. Mazza), Carolyn.stalvey@medicine.ufl.edu (C. Stalvey), steven.yale.md@gmail.com (S.H. Yale).

https://doi.org/10.1016/j.thromres.2019.06.012
Received 11 May 2019; Received in revised form 18 June 2019; Accepted 20 June 2019
Available online 21 June 2019

the Medical Subject Heading (MeSH) of the name of the eponyms and text word(s) associated with the sign.

3. Discussion

3.1. Historical aspects of venothromboembolism

Prior to Rudolph Virchow's (1821–1902) description of the three phenomenon that account for thrombosis in the pulmonary artery, physicians already began to recognize the importance of movement of blood, inflammation, and substances within blood that lead to the development of a thrombus [1–10]. It was Aristotle (384 BCE–322 BCE) who initially postulated the existence of factors in blood that promote coagulation and thrombus formation:

> There is another kind of ines or fibre that is found in the blood, but not in the blood of all animals alike. If this fibre be left in the blood, the blood will coagulate; if it be removed or extracted, the blood is found to be incapable of coagulation [11].

Virchow is credited for eloquently synthesizing these concepts, based on his own experimental work in dogs pulmonary arteries, into three principals that have stood the test of time and recalled by physicians as "Virchow's triad". These factors encompass the phenomenon of stasis of blood flow, inflammation, and hypercoagulability of blood. Virchow's name is of historic importance and most highly recognized among physicians as being associated with VTE. To provide the reader a perspective regarding his work, we briefly describe his experimental findings and contributions to this field of medicine.

Galen originally introduced the word "thrombosis" which was Greek for "curdling" [12]. However, Virchow is credited for first coining the term thrombosis to describe a blood clot when editing *Gesammelte Abhandlungen zur Wissenschaftlichen Medicin* (Handbook of Special Pathology and Therapy) collated in 1910 into a single work entitled *Thrombosis und Embolie* (Thrombosis and Emboli):

> When I was editing the *Handbook of Special Pathology and Therapy* (vol. 1., p 156), I worked on the section of obstruction and grafting in the vascular system, and decided to introduce a new name. I chose the name thrombosis, as a way to have satisfied the medical terminology [13].

Virchow was also the first to use the term "embolia" when describing the processes whereby a loose body or thrombus is propagated in the blood stream:

> For the question naturally arises how far particular disturbances that can be designated by the name pyaemia may, in the consequence of softening the thrombi, be evoked in the body. To this we may answer that secondary disturbances certainly frequently occur, but not so much by the immediate introduction into the blood of the liquefied softened masses as by the detachment of larger or smaller fragments from the end of the softening thrombus which are carried along by the blood current and driven into remote vessels. This gives rise to the very frequent process upon which I have bestowed the name *Embolia* [14].

He reported based on autopsy studies that pulmonary emboli arise from venous thrombi:

> It remains to be explained how primary spontaneous coagulation of blood occurs in the pulmonary artery, since this mode of genesis has been established as a fact by eminent investigators. Such coagulation would be quite similar to what we see occurring spontaneously in the body veins and right heart, and thus, the same conditions would have to be established for their formation. Of course, I limit myself here to the actual primary occurrence of clots, regardless of previous diseases of the lung parenchyma, or the vessel walls. I explain from the outset that I am not against the possibility of blood clotting in the pulmonary arteries at all, but only against the possibility of such coagulation developing under the same conditions, or, as one says, for the same reasons as in the veins of the body. If such a possibility existed, an argument which I have made earlier and in which I still believe would be entirely true, namely, that in all cases where old, primary clots were found in the pulmonary artery, upon closer examination, the origin in the veins can be show. Therefore, pulmonary clots never occur without venous clots and the existence of the former is sure proof of the occurrence of the latter [15].

In a series of experiments, he introduced several different foreign substances into the pulmonary artery and accounted for three possible consequences of obstruction within this artery [13]:

1) Phenomena of irritation of the vessel and its neighborhood,
2) Phenomena of blood clotting,
3) Phenomena of the interruption of the bloodstream.

Virchow also described the mechanism leading to inflammation of the vessel wall and propagation of a clot:

> My experiments on the artificial obstruction of the pulmonary arteries as well as those about the obstruction of the body's arteries have, in part, shown another way for developing disease. Emboli acting as mechanical or chemical irritating bodies in the vessel walls lead to secondary inflammation, which varies according to the nature of the stimulus and can extend far beyond its original location. (…) Already in the preceding we were repeatedly able to identify primary inflammation of the vessel wall and the consequent thrombus formation. Here we must dwell on it even more specifically, because it is precisely this form which most closely resembles the doctrinaire notions of phlebitis, arteritis, and endocarditis, and which has been studied by most investigators, without, however, finding a corresponding solution. Here arises the question of how inflammation of the vessel wall produces coagulation of blood. For we cannot allow the view that the mass that fills the vascular lumen is an exudate of the vessel wall. But as for this form of thrombosis we must reject both the view of Cruille, that the coagulation of the blood is the first effect of inflammation, and Rokitansky's statement that the exudate emerging from the wall of the vessel causes blood to coagulate. No one has seen such an exudate. Thus, it will be shown with certainty that inflammation brings its first findings not in the coagulation of the blood, but rather in the alteration of the vessel walls [13].

Therefore, a careful analysis of Virchow's work shows that his results were in reference to the pulmonary artery and embolus, and not the deep veins of the legs. His discussion and conclusions were in reference to the consequences of thrombosis and propagation of a thrombus (embolus) rather than development of a clot. He also erroneously believed that a thrombus leads to inflammation, rather than a thrombus occurs as a result of inflammation. Nevertheless, it could be inferred that by determining the consequence of a thrombus he indirectly discovered and synthesized the properties and conditions that cause VTE.

3.2. Trousseau's sign

Armand Trousseau (1801–1867) was born in Tours, Indre-et-Loire, France and received his doctorate from the Hôpital Générale, Paris in 1825 [16]. He was Associate Professor in the Faculty of Medicine, Paris in 1827, and recipient of the Legion of Honor in 1837 [17,18]. In 1839, he served as physician at the Hôpital St. Antoine followed by an appointment as Chair of Therapy and Pharmacology [16]. He was appointed Professor of the Medical Faculty and physician at the Hôtel Dieu, Paris in 1850 [19], elected member of the French Academy of

Table I
Thromboembolism eponymous signs 1873–1945.

Name	Year	Description of sign
Trousseau	1873	Patients with malignancy present with cachexia, lower extremity swelling with hardening of the saphenous and crural veins. Trousseau's sign of malignancy also known as Trousseau's syndrome refers to the presence of spontaneous recurrent or migratory venous and arterial thrombosis occurring at multiple sites (including the superficial veins) in patients with a known or occult malignancy. The other clinical feature of this syndrome includes microangiopathic hemolysis, nonbacterial thrombotic endocarditis and cardiac emboli.
Mahler	1895	During the pueperium, increased pulse rate without a corresponding rise in temperature.
Rieländer	1906	During the pueperium, pain in the groin under Poupart's ligament and pressure on the same side of the abdomen.
Michaelis	1911	During the pueperium, subfebrile rise in temperature.
Ducuing	1929	Pain located under the inguinal ligament, iliac fossa, popliteal fossa or calf elicited by gentle pressure.
Denecke	1929	Sharp pain in the sole of the foot typically located in the posterior region of the plantar arch occurring upon standing after prolonged bedrest.
Olow	1930	Calf is grasped between the knee and lower and middle third of the leg (where the triceps surae forms the Achilles tendon). Sign is positive if pain is felt when forward moderate pressure is applied anteriorly toward the tibia.
Payr	1931	Pain on pressure over the medial plantar aspect of the foot at the calcaneus inferior to the ankle. Early sign of lower extremity thrombosis being in some cases the only symptom occurring immediately or within 24 h prior to the development of edema, leg swelling, calf infiltration and increased resistance of the calf muscle upon passive dorsiflexion of the foot.
Meyer	1932	Pressure applied over the posterior tibial vein medially in a posterior to anterior direction against tibia caused pain.
Krieg	1935	Pressure applied over the vein on the adductor canal, groin, lower leg, calf or sole of the foot caused pain
Westermark	1938	Absent vascularity on chest radiograph in patients with pulmonary embolism.
Pilcher	1939	In patients with an unexplained rise in temperature, uncover both legs for 10 min. If thrombus is present the leg is warmer compared to the unaffected leg.
Hampton	1940	Irregular humped shape shadow on radiograph in patients with pulmonary infarction.
Homans	1941	Discomfort behind the knee on forced dorsiflexion of the foot.
Bisgaard	1941	Infiltration of the calcaneo-malleolar region manifested as a deep pressure pain.
Bauer	1941	Restlessness of the leg and transitory aching of the calf.
Bancroft	1945	Both knees are semiflexed with the feet resting on the bed. On the affected leg palpation reveals the presence of edema. Pain is elicited when the gastrocnemius muscle is compressed against the posterior surface of the tibia.

Medicine in 1856 [20], and served as chair of Therapeutics and Medical Matters at Charité Hospital in 1864 [16].

Trousseau recognized the occurrence of thrombi in patients with cancer [21,22] (Table I):

> A long well known fact that the blood of cachectic persons has a strong tendency to coagulate explains the frequency of coagula in the vessels of tuberculous patients who have reached the cachectic stage of their disease. I wish also to recall to your recollection some of the facts, which prove that the same spontaneous coagulation is common in cancerous patients and gives rise to phlegmasia alba dolens. In Saint-Bernard's ward, I had frequent opportunities of observing this phenomenon in women affected with cancer of the uterus. Patients of this description in the last stage of their disease present with symptoms of cachexia, and then, all at once, the inferior extremities become swollen soon after which the saphenous and crural veins can be felt to be hardened. In such cases, it is found on examination after death that their hard condition is owing to cruoric or intra-vascular fibrinous clots (…). I have long been struck with the frequency with which cancerous patients are affected with painful edema in the superior or inferior extremities, *whether one or the other was the seat of cancer*. The frequent concurrence of phlegmasia alba dolens with an appreciable cancerous tumor led me to inquire whether a relationship of cause and effect did not exist between the two, and whether the phlegmasia was not the consequence of the cancerous cachexia. I have since had an opportunity of observing other cases of painful oedema where there existed a cachexia referable neither to the tubercular diathesis, puerperal state, nor chlorosis. At autopsy I found visceral cancer but during life there was no appreciable cancerous tumor. I have thus been led to the conclusion that when there is a cachectic state not attributable to a tuberculous diathesis, or to the puerperal state, there is probably a cancerous tumor in some organ [23].

It should be recognized that another sign which bears his name is in associated with latent tetany [24] (Table II).

3.3. Mahler sign

There is limited historical information on Richter A. Mahler (1863–1941). His manuscript entitled “Thrombose, Lungenembolie und plötzlicher Tod” (Thrombosis, Pulmonary Embolism and Sudden Death) during the postpartum period, was part of a two-volume collection of papers written by physicians at the Dresden Gynecology Clinic, and edited by Professor G. Leopold, Medical Council of Obstetrics and Gynecology [25]. Cumston publication in 1908 contained a translation of Mahler's work (Table I):

> In a typical thrombosis the pulse rises and continues to rise while the temperature remains perfectly normal. If there is edema, a thrombotic strand may be palpable, or, if pulmonary symptoms arise the pulse beat reaches its highest point and occasionally the temperature goes up at the same time. While the temperature will be found to fall during the next few hours, the pulse will remain rapid for several days. Sometimes the recognition of thrombosis is made more difficult by a gradual increase in the pulse rate simultaneous with a slight rise in temperature at the very commencement of the process. In such cases however, the diagnosis is pretty certain when there is a marked divergence between the pulse and temperature curves. On the other hand, there are cases where the gradual increase in the pulse rate is not marked, the temperature is normal, and then, again, there may be a small and very rapid pulse. In these cases one should also take into consideration the possibility of a thrombosis [26].

Mahler believed that extensive thrombosis within the pulmonary circulation leads to an increased cardiac output and more frequent and weaker contractions of the heart, and that the pulse increases proportionally to the extent of resistance caused by size of the thrombus and decreases with the formation of collateral circulation [27]. He found that the pulse increases disproportionate to the temperature; after an initial increase, the temperature falls while the pulse steadily increases [28].

3.4. Rieländer sign

Wilhelm August Rieländer (1875–1926) was born in Erfurt, Germany, and graduated from the School of Medicine in 1899. After serving as a voluntary assistant for two months in 1900, he was subsequently appointed resident in the gynecology clinic at the University Women's Hospital in Marburg, Germany [29]. Between 1903 and 1911, he served as a head of the training of midwifery students, and in 1904 received his habilitation in Midwifery and Gynecology [29]. He became

Table II
Other eponymous signs.

Name	Description of sign
Trousseau	Trousseau sign of latent tetany. Spasmodic contraction of the hand and digits. The fingers are flexed at the metacarpalphalangeal joint and extended at the proximal and distal phalangeal joints with the thumb is flexed and adducted into the palm.
Payr	Payr sign of the knee is an increase pain in the medial knee when downward pressure is applied to the knee while the patient is sitting. Found in patients with disease involving the medial meniscus and its posterior horn

Table III
Location of symptom and signs.

Location	Name of sign
Inguinal ligament	Rieländer, Ducing
Thigh	Krieg
Popliteal	Ducuing, Homans
Leg	Ducuing, Meyer, Krieg, Pilcher, Bauer
Calf	Ducuing, Olow, Krieg, Bancroft
Foot	Denecke, Payr, Krieg, Bisgaard

chief physician at a field hospital during World War I, and retired as a second midwife teacher in 1920 [29].

Rieländer reported that “pain in the groin, especially under Poupart's ligament” along with “pain to pressure on the corresponding side of the abdomen” is a symptom found in thrombosis of the leg during the puerperium period [30] (Tables I, III).

3.5. Michaelis sign

We were only able to identify limited historical information on Hans Michaelis who served as a gynecologist in Königsberg, Prussia (current Kaliningrad, Russia) [31]. He described in his paper titled “Prodromalerscheinungen der puerperalen und postoperativen Thrombose und Embolie” (Prodromal signs of puerperal and postoperative thrombosis and embolism) the relationship between temperature and thrombosis:

> If I were asked how do you explain these small temperature increases? The expression of what kind of process should they be attributed?, I would say it is a sign that a thrombus is there, which is not organized in the normal way, but is perhaps in the process of dissipating from which even the smallest particles are already crumbling and circulating in the blood. However, without any bacterial involvement the softening can be explained by the increase in temperature. (…) I do not claim that any unexplained occurrence of subfebrile temperatures indicates an embolic risk, but I must say again according to my observations puerperal postoperative thromboses and embolisms do not occur out of the blue; there is a premonitory symptom in the form of subfebrile temperature. If this occurs, one must think of the possibility of thrombosis or embolism and act accordingly [32].

Michaelis believed that the temperature is altered prior to thrombosis and embolism. According to Michaelis “not every subfebrile temperature is an index of the onset of thrombosis, but anything over 37.5 °C suggests the necessity for caution and close supervision of the patients, guarding them against any violent movement, any excitement or fright, and keeping the bowels loose with oil injections, a mild laxative, and suitable diet.” [33] (Table I).

3.6. Ducuing sign

Joseph Ducuing (1885–1963) was born in Toulouse, France, and received his Laureate from the Faculty of Medicine, University of Toulouse in 1912 [34]. At the Hôpitaux de Toulouse (Toulouse Hospital), he was appointed Chief Operating Physician in 1919, associate professor of general surgery in 1920, and Professor and Chief Surgeon at the Faculty of Medicine in 1922 [34]. In 1929, he was selected as Director of the Anticancer Centre (Centre Claudius Regaud) and founded the regional association for the fight against cancer in 1932 [34]. He was appointed chair of the surgical clinic of cancer from 1938 to 1940 [35]. In 1946, he served at the Purpan Hospital [34], and in 1952 was appointed President of the French Association for Cancer Studies [36]. The Warsaw Hospital was renamed “The Joseph Ducuing Hospital” in 1979 in his honor [34]. He recorded in his book *Phlébites Thromboses et Embolies Post-Opératoires* (Phlebitis, Thromboses and Postoperative Embolism) the finding that:

> [p]hlebitis of the leg causes spontaneous pain in three main regions, inguinal, popliteal, and the calf. This pain is most often mild, but it is sometimes severe, deep, dull, gravitational, and continuous, with some intermittent twinges lasting a few days and gradually improving. We have again seen spontaneously painless postoperative phlebitis with pain expressed by gentle pressure on the groin, the popliteal fossa, or the calf. We insist in particular on a small pain sitting behind the inguinal ligament or in the iliac fossa that we systematically look for in all our patients. This pain is of great diagnostic value to us especially when it is located on the left side or when it is associated with either a very slight ballooning of the abdomen, a shiny and edematous condition of the skin, or a slightly exaggerated circulation of the veins [37].

There is no consensus in the literature what constitutes Ducuing sign. It has been described in relation to ballottement or a back and forth movement of the calf [38–41]. Other authors described Ducuing sign in reference to pain on compression or palpation of either the calf or groin [42–44] (Tables I, III).

3.7. Denecke sign

Kurt Denecke (1903–1991) studied medicine at the Universities of Göttingen, Würzburg and Munich, Germany passing his state medical examination in 1927 [45]. He served as First Senior Physician at the Surgical University Clinic, Erlangen Germany in 1948, a position which he formerly held prior to WWII in 1937 [45]. In 1952, he was appointed Associate Professor at the University Hospital [54]. Denecke was acting director of the clinic in 1954, and Chief Physician of Surgery at the Surgical Clinic, City Hospital Fürth, in 1956 [45].

While serving as Professor of Surgery and Chief Physician at Klinikum Fürth, Denecke described the finding of lower extremity venous thrombosis in his paper entitled “Der Plantarschmerz als Frühsymptom einer beginnenden Thrombose der unteren Extremität.” (Plantar pain as an early symptom of incipient lower extremity thrombosis):

> It has been observed that in certain cases, patients who were bedridden for a long time and accidentally get up will experience a sharp pain in the soles of the feet. This pain was usually localized to the posterior area of the plantar arch. Strikingly about 24 hours later the clinical symptoms of lower leg thrombosis occur in the affected leg [46].

As to the pathogenesis of the pain, Denecke hypothesized that flexion of the foot causes entrapment of the nerve or pressure on the vascular and nerve bundles leading to ischemia or congestion and

dilation of the vessels in the nerve bundles. [46]. Hence, he believed that the pain is caused by mechanical mechanisms initiated by foot flexion. He found that plantar thrombosis was found in 2 of 22 cases where there was no clinical evidence of thrombosis of the leg. Conversely, plantar thrombosis was identified in 7 patients all of whom who had clinically diagnosed femoral vein thrombosis. He recommended that patients with suspected lower extremity thrombosis should routinely be assessed for pressure pain on the medial half on the dorsum of the foot, immediately distal to the toes [46] (Tables I, III).

3.8. Olow sign

John Olow (1883–1948), was born in Malmö, Sweden and received his medical license in 1910 and doctorate of medicine in 1913 from Lund University [47]. In 1914, he held a clinical teaching position in obstetrics and gynecology and in 1917 served as first-class marine physician [47]. He was appointed Professor of Obstetrics and Gynecology at the Upsala, Sweden, Gynecological Clinic and Senior Physician in the Obstetric and Gynecology Department at Academic Hospital in 1923 [47]. Olow served as Professor of Obstetrics and Gynecology at the Karolinska Institute in Stockholm and Chief Physician and Director at the General Maternity Hospital in 1931 [47]. He described a maneuver for detecting thrombosis of the leg:

> The calf is grasped below the knee between the lower and middle third of the leg, where the triceps surae muscle forms the Achilles tendon. Forward moderate pressure is applied against the tibia. Thrombus is present if the patient feels pain. (…) In general, this sign is present from a few hours to a few days before the appearance of the classic symptoms of thrombosis. However, in some cases, the sign is ephemeral, and so, if we examine only once or twice, we can miss it; if you repeat the exam once or twice a day, it almost always appears on one occasion or the other [48].

Olow believed that this was a useful early sign for detecting DVT of the leg (Tables I, III).

3.9. Payr sign

Erwin Payr (1871–1946) was born in Innsbruck, Austria in 1871 and completed his medical training at the University of Innsbruck in 1894 [49,50]. That same year, he served as an Assistant in Internal Medicine and Surgery at the Institute of Pathological Anatomy in Vienna [49,50]. Until 1906, he was a primary physician in the Surgical Gynecological Department at Graz Municipal Hospital and was subsequently appointed chair, Department of Surgery, at the University of Greifswald Paul Leopard Friedrich in 1907 [49,50]. He was appointed Ordinarius Professor of Surgery in Königsberg (Prussia) in 1910 as well as in Leipzig from 1911 to 1936 where he also served as Director of the University Clinic [49,51]. Payr made a number of extraordinary contributions involving multiple therapeutic areas in medicine and surgery. His surgical expertise spanned the fields of orthopedics, neurosurgery, organ transplantation, abdominal, urogenital, otolaryngology, and thyroid surgery [52–67].

Gerhard Tschmarke, an Assistant at the Surgical University Clinic of Leipzig under the auspices of Payr as director, reported in their paper entitled “Erfahrungen über den Fußsohlendruckschmerz als Frühsymptom der Thrombose” (Experience of foot sore pressure pain as an early symptom of thrombosis) that in 152 postoperative cases, thrombosis was found in 21 (13%) patients. Pain in the sole of the foot was the first among other signs found in 5 of 16 cases and in the remaining was detected simultaneously with the appearance of mild subjective or objective signs of thrombosis. The sign was absent in 5 cases of thrombosis [68,69]. He believed that thrombosis in the lower extremity begins at the rete plantar venosum located in the plantar aspect of the feet which contains particularly large numerous vessels [69]. In the description of the physical finding Payr stated (Tables I, III):

> Since I have been careful, I find more and more often people with otherwise uncertain pictures of thrombosis and in those with small pulmonary embolism without a clear area of origin of the clot, a noticeable pressure sensitivity on the inside of the sole of the foot from the heel bone just below the inner ankle. A little later, the area of the poster tibial vein between the tibia and achilles tendon is sensitive to mild edema and patients sometime complain of pressure sensitivity of the heel. Passive dorsiflexion of the foot becomes painful either alone or simultaneously with pressure on the deep calf veins. On closer examination, however, the point of pain is most pronounced at the location described. (…) This foot pressure soreness is a “warning” sign of a developing thrombosis, and may indicate a threatening embolism. (…) I find these early symptoms of deep vein thrombosis very important. (…) In the absence of any objective sign of thrombosis one will rarely be inclined to add weight to the patient's information about a sensation of tingling or numbness in the foot, tension in the calf, or heaviness in the lower leg. However, if this information occurs simultaneously with positive evidence of foot pressure soreness then one is more likely to be prepared for the occurrence of thrombosis, and not be surprised if within 24 hours or later, the first flawless objective signs of thrombosis are detectable. Foot pain in the sole of the foot is therefore indeed an early sign of the thrombosis developing in the area of the lower leg [70].

It should be recognized that another sign, which bears his name, is associated with diseases involving the knee (Table II).

3.10. Meyer sign

There is little historical information identified about Otto Meyer (1896-) who practiced medicine in New York City. As a well-known specialist of phlebitis and its complication, he published the book entitled *Phlebitis*: *The Hidden Cause of Most Leg and Foot Ailments* in 1940 [71]. In 1932, Meyer described in his paper entitled “Symptomenkomplex und Therapie des Ulcus phlebiticum” (Complex symptoms and therapy for phlebitic ulcers):

> The best pressure and touch point for the posterior tibial vein seems to be on the medial side of the tibia in the lower third, when pressure is applied from below against the tibia, pushing the vein against the bone [72].

Meyer identified an area, located over the posterior tibial vein for best detecting phlebitis of the leg (Tables I, III).

3.11. Krieg sign

Erich Krieg (1902–1970) was born in Germany. He was elected the first President of the German Association for Phlebology (later German Society for Phlebology) and served as chairman of the society whose goals were to train, develop theory and standards for physicians practicing phlebology [73]. In 1959, he was elected the first President of the International Union of Phlebology [73]. Since 1975 the Erich Krieg Prize has been given to scientists whose work in phlebology serves to advance science and practice [73]. Krieg in his description of venous thrombosis reported that:

> The most valuable early sign is pressure pain in the vein. It can clearly be detected up to 24 hours prior to the appearance of clear signs of thrombosis. It occurs on the thigh mostly in the area of the adductor slit or in the groin, on the lower leg, calf or sole of the foot [74].

Krieg believed that this is a useful early sign for detecting thrombosis of the leg in cases of inflammatory changes within the vein caused by infectious diseases (Tables I, III).

3.12. Neumann sign

We have purposely omitted the name Robert Neumann (1902–1962) from the Table I containing eponymous names due to the atrocities he committed against humanity in the concentration camps as a member of the Schutzstaffel (SS), a paramilitary organization under the Nazi party [75–77]. We have rightfully stated that medical eponyms are intended to be honorific term(s) used to acknowledge the person(s) whom the discovery is attributed. There use should be reserved and restricted to honor only those individuals who uphold the principles of the Hippocratic oath-*primum non nocere* or "first, to do no harm". The honor should not be bestowed upon an individual, regardless of his/her other altruistic lifetime accomplishment(s), to someone who intentionally harmed others by committing research under unethical circumstances, or who was involved in atrocities against humanity. Thus, the eponymic sign ascribed to Neumann is ethically inconvenient to be used in medical discourse.

Robert Neumann was born in Nüssdorf, Silesia and received his medical degree in medicine and musicology at the Universities of Wroclaw and Hamburg, Germany in 1929 and doctorate from the University of Hamburg in 1930 [76]. In 1932, he was appointed Assistant, in 1934 senior physician, and in 1935 director at the Pathology Institute of the Robert Koch Hospital in Berlin [77]. He received his habilitation and qualified as a privatdozent (name assigned to a habilitated scientist) in 1936 [76]. He joined the SS in 1933, was SS-Untersturmführer (second lieutenant) between 1936 and 1937, and promoted to SS-Obersturmführer (first lieutenant) in 1938 [76–78]. He was also a member of Lebensborn e V (Nazi hygiene and health ideological practices) as well as the National-socialist German Worker's Party (NDSAP) in 1937 which propagated anti-semitic and nationalism beliefs [78]. In 1940, he established the Pathological Institute at Tongji University in Shangai, China, a mission established for "cultural-political reasons", and served as its director in 1941 [77]. From 1945 to 1948 he was retained as an allied prisoner [75,77], and in 1948 worked as a scientific employee for STADA pharmaceutical company, Tübingen, Germany, later serving as head of the hospital in Reutlingen, Germany [78].

He identified in 163 autopsies of the lower extremities that thrombosis was identified in 100 patients. The lower leg was involved in 87% of these patients with the second most common site being the plantar vein in the foot (71%). He found that the malleolar veins, which connect the plantar from the lower leg veins, were infrequently thrombosed. Thus Neumann postulated that thrombi originate more commonly in the calf and less frequently in the plantar region. Based on their point of origin, he described a benign and malignant type of venous thrombosis. The benign type originates in the calf and is characterized by an increasing frequency with advancing age, slow progression, and multiple non-fatal pulmonary emboli. The malignant type originates in the plantar veins, occurs early in life, rapidly progresses and ascends to the femoral veins, and is more likely to cause fulminant lethal pulmonary emboli [79].

He proposed that thrombi occur based on the concept of coherent segmentation. The segmentations occur at points where the vein is crossed-over by ligaments, tendons, and bone. Two crossovers named "proximal" and "distal" plantar vein points, caused by segmentation of the veins in the plantar region, are responsible for lateral plantar vein thrombosis. He believed that their identification is important for the early detection of the malignant type of venous thrombosis. The "distal foot pressure pain" was postulated to occur even earlier than the "proximal plantar pressure pain." He further emphasized that people with flat arch (pes planus) are more likely to develop segmented thrombus in the lateral plantar vein compared to those with a normal or high arch (pes cavus) [79].

He described the method for detecting these points: "The proximal plantar vein point is easy to find; it lies close to the malleolus internus next to the calcaneus." [79]. To identify the distal point:

> The patient is ideally placed in a prone position. The sole of the foot must - which is important - be at a right angle to the longitudinal axis of the lower leg. The greatest width of the sole of the foot above the heel and over the tarsal bones are divided in half, and the two half points of the plantar skin are connected by a line. Then the medial highest points of the internal malleolus and the medial heads of the metatarsals are marked by crossed lines. A ruler is placed at these two points, the distance of these two points is measured, and at the point of bisection of this line the vertical is established (by applying a straight rod or pencil to the half point and the medial side of the foot). The distal plantar vein point lies where this perpendicular intersects the halo line of the sole of the foot [79].

He believed that the "foot pressure pain" of Payr as described by Tschmarke is a very sensitive sign of early thrombus in the foot and calf and is located exactly after the "proximal plantar venous point." He noted that the "distal foot pressure pain" may appear even earlier than the "proximal plantar pressure pain". He called for further investigation to confirm his findings [79].

3.13. Westermark sign

Nils Johan Hugo Westermark (1892–1980) was born in Stockholm, Sweden and received his medical degree in 1919 from Karolinska Institute School [80,81]. He was a sailor representing Sweden on the boat Sans Atout, when he competed in the 1912 Summer Olympics, winning the silver medal in the 8-meter class [82]. He served in the department of radiology in Sabbatsbergs Hospital (1920–1921), Karolinska Radium Hospital (1922–1923), St. Eriks Hospital (1922–1929), and St. Görans Hospital (1930–1957) [90]. He received his PhD in radiology in 1930 and was appointed Associate Professor at Karolinska Institute that same year, followed by the title of professor in 1955 [81]. He served as Secretary General of Nordisk Förening för Medicinsk Radiologi (Northern Association for Medical Radiology) in 1933 [83], and between 1946 through 1947 delivered a series of lectures at the University of Minnesota at the Center for Continuation Study on thoracic radiology compiled into a textbook entitled *Roentgen Studies of the Lung and Heart* [84].

Westermark published in 1938 "On the roentgen diagnosis of lung embolism" the radiographic appearance of embolism of the pulmonary artery in patients without pulmonary infarction showed:

> (…) ischaemia of the distributed area of the pulmonary artery on the peripheral side of the embolus or emboli has appeared on the radiogram as a local and well defined clarified zone with diminished or absent vascularization. In an area corresponding to the site of the embolus and on the central side of this the vascular design has been well maintained but seems to stop abruptly. (…) It is clear, therefore, that lung embolism without infarction leads to anemia and diminished vascularization and not hyperaemia and increased vascularization as assumed by previous authors such as Assmann and Frimann-Dahl [85].

In the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study, 1063 patients with suspected pulmonary embolism based on chest radiography received a pulmonary angiography. Pulmonary embolism was confirmed in the right and left hemithoraces in 259 and 233 patients respectively and excluded in 680. Westermark sign had a low sensitivity (14% and 8%) but high specificity (92% and 96%), in the right and left hemithoraces for the diagnosis of pulmonary embolism. The positive predictive value was 38%. It was found to have a positive predictive value of 38% and a negative predictive value of 76% [86]. Thus, the presence of this radiographic finding suggests the presence of pulmonary embolism, however, its absence does not exclude the diagnosis (Table I).

3.14. Pilcher sign

Robin Sturtevant Pilcher (1902–1994) was born in Middlesex, England and received his medical degree at the University College of London in 1927 where he subsequently spent his entire medical career. In 1938, he was appointed professor of surgery and from 1938 to 1967 Director of the Professional Surgical Unit [87]. He served as Chairman of the Court of Examiners of the Royal College of Surgeons in 1965 and in 1967 was appointed Fellow of the Royal College of Physicians (FRCP) [87].

Pilcher reported that thrombosis of the leg is diagnosed based on the findings of delayed cooling on exposure, pain, tenderness, slight fullness, and cyanosis of the superficial veins. The delayed cooling test is performed as follows:

> A patient has an unexpected rise of temperature and the legs are exposed for ten minutes, even if they look normal. A marked difference of temperature is suggestive of thrombosis, the thrombosed leg being warmer, and I do not remember any case showing this sign alone which did not subsequently develop others [88].

He emphasized, “This test is vitiated by operation or inflammatory lesions of the limb in question” [88] (Tables I, III).

3.15. Hampton sign

Aubrey Otis Hampton (1900–1955) was born in Copeville, Texas, received his medical degree from Baylor University Medical School, Texas in 1925, and radiology training at the Massachusetts General Hospital, Boston, Massachusetts in 1926 [89]. He was appointed chief of the Department of Radiology at Massachusetts General Hospital in 1941 and subsequently Chief of Radiologic Service at Walter Reed Hospital from 1942 to 1945 [89,90]. After World War II, he led the Department of Radiology at Garfield Hospital, Washington, D.C. and served as a radiology consultant at Walter Reed Hospital [90,91]. He was than appointed chief consultant and medical director of radiology of the Veteran's Administration [89] and created a radiologic pathology fellowship at the Armed Force Institute of Pathology [89]. Hampton and Benjamin Castelman described the radiographic shape of a pulmonary infarction:

> The medial or cardiac margin of an infarct is convex toward the heart, presents a serrated margin, and, if the central roentgen ray is tangent to it, a convex, slightly irregular “hump”-shaped shadow is produced. When the lateral costophrenic margins of the lung are involved, the posteroanterior view of the chest shows the “hump”-shaped shadow. When the anterior and posterior costophrenic margins are involved, the “hump” shadow is seen only in the lateral view. (…) The cardiac margin of the infarct is rounded or “hump”-shaped instead of pointed [91].

The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study evaluated 1063 patients with suspected pulmonary embolism (PE) based on chest radiography, whereby PE was confirmed angiographically in the right and left hemithoraces in 259 and 233 and excluded in 680 patients. A sensitivity of 22% and 24% and specificity of 82% was found for pulmonary embolism in the right and left hemithoraces respectively. The positive predictive value for the right and left hemithoraces was 29% and 27% respectively [86]. The presence of Hampton's sign suggests the presence of pulmonary infarct, but its absence does not exclude the diagnosis (Table I).

3.16. Homans sign

John Homans (1877–1954) was the fifth generation of physicians in his family to have received his undergraduate education at Harvard University in 1899, graduating from Harvard Medical School as class valedictorian in 1903 [92,93]. He completed a surgical internship at the Massachusetts General Hospital, and served as an assistant to Maurice Richardson, Mosely Professor of Surgery at Harvard from 1903 to 1908 [94]. He received further training under the aegis of Dr. Cushing at Johns Hopkins University School of Medicine, Baltimore, on experimental hypophysectomy in the Hunterian Laboratory from 1908 to 1909. Cushing, then surgeon in chief at the Peter Bringham Hospital in 1910, appointed Dr. Homans in 1912 as one of his principal associates [93]. In 1946, he retired as clinical professor of surgery, emeritus.

Throughout his career he published multiple scientific manuscripts, including the first edition of *A Textbook of Surgery* and the monograph in 1931, and *Circulatory Diseases of the Extremities* in 1939. He was a founding member of the Society of Vascular Surgery [92]. For his accomplishments throughout his career, he was honored by his name being bestowed - John Homans Chair of Surgery at Harvard Medical School and the John Homans Lectureship in the Society of Vascular Surgery (SVS) in 1950 [95].

Among his many accomplishments, he was the first to advocate for ligation of the femoral vein in the groin below the sphaphenofemoral junction to prevent pulmonary emboli [92], and contributed extensively to the pathogenesis and treatment of lymphatic and venous ulcers in the legs [93]. Homans described the dorsiflexion sign in 1941 in his paper “Exploration and division of the femoral and iliac veins in the treatment of the thrombophlebitis of the leg” as “discomfort behind the knee on forced dorsiflexion of the foot.” [96]. In describing the course of disease he wrote:

> The swelling and cyanosis, if any, always disappear in a few days, but the dorsiflexion sign usually lasts for a week or more. (…) If, on the other hand, when the patient first gets up, the discomfort, edema and especially the dorsiflexion sign reappear, the superficial femoral vein is at once exposed and divided [96].

He further stated that, “This type of thrombosis, though difficult of identification, can often be diagnosed, whether or not embolism has occurred, by a combination of clinical symptoms with discomfort behind the knee on forced dorsiflexion of the foot” [96].

Allen et al. in their study of 202 patients treated by femoral vein interruption at Massachusetts General Hospital coined the term “Homans sign” for his namesake who first described it. In a footnote they said, “Dr. John Homans calls it the dorsiflexion sign. It is carried out by dorsiflexion of the foot with the knee straight. If the patient complains of pain or soreness in the calf muscle when this is done the sign is interpreted as positive for venous thrombosis” [97]. In 1944 in his paper “Diseases of the veins” commenting on Allen et al. findings, Homans said:

> I prefer to call it the dorsiflexion sign,-this was found to be present in 42 per cent. I regard this dorsiflexion sign as having a greater degree of importance than would be judged from this analysis. Actually, dorsiflexion of the feet is intended to bring out, on the side of the venous thrombosis, some degree of irritability of the posterior muscle, the soleus and gastrocnemius. Discomfort need have no part in this reaction. Dorsiflexion may be less complete in response to an equal degree of upward pressure on the affected side as compared with the normal, or the patient may involuntarily flex the knee as the forefoot is forced upward, to release the tension on the posterior muscle. If one looks on the dorsiflexion sign as evidence of even the faintest irritability of the posterior muscle, the early stage of the thrombosis occurring with and about them,-the sign will probably be found present more frequently than either tenderness or swelling [98].

Thus, Homans original description of a positive test included “irritability” of the calf muscle, incomplete dorsiflexion of the foot, and/or involuntary flexion of the knee upon forced dorsiflexion of the foot. It is historically interesting that Tschmarke in his 1931 paper reported an objective sign of existing thrombosis of “[i]ncrease resistance of the calf musculature against passive dorsiflexion of the foot” [69].

Several textbooks and manuscripts when describing Homans sign stated that the knee should be in flexed, in a slightly flexed position, or at a 30° angle [99–104]. Other authors used the term abrupt or sudden when performing ankle or foot dorsiflexion with pain occurring in the calf or popliteal region [105–110]. As delineated by Homans, discomfort (slight pain) is not required for a positive test, occurs behind the knee, and irritability involves the soleus and gastrocnemius muscles [98].

Homans, despite his objection assigning his name and limitations of this sign, undoubtfully remains the most widely recognized eponym attributed to venous thrombosis of the calf. The wide range of sensitivity and specificity found in Homans sign is attributed to a number of factors including conditions in the leg that cause a false positive rate such as tenderness of the calf, muscle, fascia, nerves, and subcutaneous tissues such as herniated intervertebral lumbar disk, Baker's cyst, calf muscle spasm, and shortened heel cord as well as not performing the sign as originally described, thus limiting the interpretation and conclusions of this sign [108,111] (Tables I, III).

3.17. Bisgaard sign

There is limited historical information about Holger Ove Bisgaard (1880–1943). He was born and lived in Glamsbjerg, Denmark [112]. Inscribed in his tombstone are the words “Many were cured. He gave back the joy of living,” in reference to his method for treating a venous leg ulcer [112].

Bisgaard in his book entitled *Ulcus und Eczema cruris*, *Phlebitidis sequelae usw* (Ulcus and Eczema Cruris, Phlebitidis Sequelae, etc.) described the treatment of a venous leg ulcer using a combination of leg elevation, elastic support combined with deep firm massage around the ulcer. He identified early findings found in patients with lower extremity phlebitis and in those who develop venous ulcers of the lower extremity caused by venous insufficiency and stasis:

> [i]n the lower extremities of patients who are bedridden or when dealing with elderly persons, abnormalities of some kind can often be demonstrated. There may be infiltrations in the muscles that cause rheumatoid-type pain, stiffness of the joints and infiltration in their environment, but the most common anomaly is undoubtedly the infiltration of the calcaneo-malleolar region, which I have for the sake of simplicity would like to call it a ‘backdrop’. From here it often spreads out into the neighboring regions especially towards the back of the ankle. (…) The infiltration of the setting, which appears to be present in virtually every ulcer or eczema of this region cannot always be seen, and not always felt, but always manifests itself as a deep pressure in this region. (…) This provokes the pathognomonic symptom of this condition; a facial expression that lies approximately in the middle between smiles and weeping [113].

Bisgaard described the etiopathogenesis for the infiltration of the ankle:

> The acute onset of infiltration is seen in debilitating conditions of various kinds such as after birth and surgery, and often as a precursor to phlebitis or phlegmia, and presumably as a direct cause. This acute infiltration should never be underestimated; on the contrary, it should be accorded the greatest importance as the first source of many embolisms, and as long as this is still easily possible eliminated by massage and intensive movement of the ankle joint (…).
> This is based exclusively on clinical observations that I have made in many years on a large number of patients. It may be justified if I point to the new pathological -anatomical examinations (Rössle, Voegt, Neumann, Frukholm, et al.) that find thrombosis in exactly the same regions (thigh, ankle, and calf) where I found infiltrations [113].

Bisgaard recommended that vigorous massage and mobilization was the best method to prevent thrombosis and embolism (Tables I, III).

3.18. Bauer sign

Gunnar Bauer (1895–1970) graduated from medical school from the University of Lund and Karolinska Institute, Stockholm, Sweden [114]. He completed his training in cardiothoracic surgery at Sabbatsberg and Malmö Hospitals in Sweden [115]. He spent several years in Sveg, Northern Sweden, followed by service as Chief-Surgeon at Mariestad General Hospital in Mariestad, Sweden where he spent the remainder of his career [114]. He made significant contributions that led to a better understanding of DVT by applying the technique of standing descending venographic studies in patients with acute thrombosis and venous incompetence of the legs. He also pioneered the use of heparin to treat VTE and promoted early ambulation to prevent post-thrombotic syndrome [114]. Bauer's venographic work paved the way to an enhanced knowledge of the evolving thrombotic and post-thrombotic syndrome. He demonstrated that thrombosis of the lower extremity starts in the calf veins and the post-thrombotic syndrome was dependent upon the patency of the popliteal and femoral venous valves [114]. He also distinguished post-thrombotic deep venous reflux from idiopathic valve incompetence. He and Knut Haeger in 1963 formed the Soceitas Phlebologica Scandinavica [115] at Mariestad, Sweden and described the symptom of restlessness and tenderness of the calf muscle as an early sign of DVT. According to Bauer:

> Heed must be given to the slightest unexplained rise in the pulse rate or the temperature, to any unmotivated general restlessness, to the least signs of pulmonary infarction and to the faintest signs of a pathologic condition of the legs, such as a transitory aching in the calf, lasting perhaps only a few hours, a slight change in the color of the skin, a small increase in the firmness of the calf muscle or a tender spot, found only with difficulty, on the back of the lower part of the leg [116].

Thus, Bauer sign refers to the general restless appearance of the patient with DVT (Tables I, III).

3.19. Bancroft sign

Frederic Wolcott Bancroft (1880–1963) graduated from Johns Hopkins University Medical School in 1906 [117–119]. In 1914, he served as Assistant Surgeon and 1916 Instructor of Clinical Surgery at Columbia University College of Physicians and Surgeons, New York City [120]. He was appointed Chairman of the Committee on Fractures of the American College of Surgeons in 1922 [121] and from 1922 to 1946 as Associate Professor of Clinical Surgery at Columbia-Presbyterian Medical Center. From 1924 to 1926 he served as Director of Surgery at Lincoln Hospital, New York City and from 1926 to 1936 Surgical Director Fifth Avenue Hospital, Associate Professor of Clinical Surgery at Columbia University, Attending Surgeon at the United States Veterans Hospital No: 81, and Consulting Surgeon at Lincoln Hospital [117,122]. From 1926 to 1946 he was Professor of Clinical Surgery at the Columbia University College of Physicians and Surgeons and Professor of Clinical Surgery at the New York Medical College from 1947 until his death [117]. In 1936, he was elected to the Board of Governors of the American College of Surgeons for a three-year term [123], and was Surgical Director of the New York City Hospital from 1936 to 1946 [117]. His expertise spanned the fields of vascular and orthopedic surgery. He was the founding member of the American Board of Surgery [124].

In his paper on “Proximal ligation and thrombectomy for phelebothrombosis of the femoral and iliac veins,” Bancroft reported that:

> Homans has described the sign, which is now given his name; if the foot is flexed dorsally it compresses the gastrocnemius muscle, and pain is experienced in the calf. I have also found that if one

semiflexes both knees, with the feet resting on the bed, the calf of the affected side gives one the feeling of a deep edema, and, as the gastrocnemius muscle is compressed against the posterior surface of the tibia, pain is elicited [125].

Thus, Bancroft described a method for detecting DVT with the patient supine and knees in a semi-flexed position (Tables I, III).

4. Conclusion

The use of eponymous terms probably cannot be avoided in the scientific literature and in daily patient care, but when cited should be appropriately used as originally described to avoid misrepresentation and to ensure their appropriate application. The physical examination is limited in some cases in its ability to identify DVT because of a reduced or absent inflammatory infiltrate, collateral circulation, and partial occlusion of the vessel. Surprisingly, with the exception of a few signs, there is a paucity of studies that assessed their accuracy and reliability during the physical examination. Furthermore, a variety of methodological limitations affect the interpretation of studies that evaluated some of these signs including a small sample size, misinterpretation of a positive test, not describing the method of performing the sign, improperly performing the test as originally described and intended, testing in a population without a validated control group, or retrospective study [110,126,127]. Thus, the accuracy of these signs in clinical practice is unknown. Further studies are required to assess the validity of these signs in clinical practice.

Funding source

The research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

[1] A. Paré, The works of that famous Chirurgion Ambrose Parey, in: Of Ulcer, Fifula's and Hemorrhoides, Lib 13 (1649) 354.
[2] R. Wiseman, Severall Chirurgicall Treatises, E. Flesber and J. Macock for R. Royston. Bookfeller to His Moft Sacred Majesty and B. Took at Ship in St. Paul's Church-yard, Anne Dom, London, 1676.
[3] J.M. Forrester, Malphighi's De Polyp Cordis: an annotated translation, Med. Hist. 39 (1995) 477–492.
[4] J. Hunter, Observations on the Inflammation of the Internal Coats of Veins, Tr. Soc. Improve. M. & Chir. Knowl, London, 1793, pp. 18–29.
[5] A. Haller, A, First Line of Physiology, W. Cullen (trans), C, Elliot, Edinburgh, 1786.
[6] M. Baillie, Of Uncommon Appearances of Difeafe in Blood-veffels, Tr. Soc. Improve. M. & Chir. Knowl. London. (1793) 119-137.
[7] J. Hodgson, I.V. Part, On the diseases of veins, inflammation of the veins, A Treatise on the Diseases of Arteries and Veins Containing the Pathology and Treatment of Aneurisms and Wounded Arteries, Dublin, 1815, pp. 511–517.
[8] W. Hewson, Properties of the blood. Of the separation of the serum; the colour of the crassamentum; and of the causes of the coagulation of the blood, in: George Gulliver (Ed.), The Works of William Hewson, F.R.S, Printed for the Sydenham Society, London, 1746, pp. 1–30.
[9] G. Andral, Essai d'Hématolgie Pathologique. (Essay on Pathological Hematology), Dublin, J. M. & Chem. Sc. 23 (1843) 288–299.
[10] C. Rokitansky, Abnormal conditions of the veins. in: W.E. Swaine, E Sieveking, C. H. Moore, G.E. Day (Eds.). (translated from the last German edition) A Manual of Pathological Anatomy Volume 4, Printed for the Sydenham Society, London, 1852 pp. 235-276.
[11] Aristotle, The History of Animals. The Complete Works. Book 3 Part 6. Translated by D'Arcy Wentworth Thompson, Clarendon Press, Oxford, 1910.
[12] S.T. Anning, The historical aspects of venous thrombosis, Med. Hist. 1 (1957) 28–37.
[13] R. Virchow, Phlogose und Thrombose in Gefässsystem. Gesammelte Abhandlungen zur Wissenschaftlichen Medicin, AM Verlag von Meidinger Sohn & Comp, Frankfurt, 1856.
[14] R. Virchow, X. Lecture, Purulent Cysts in the Heart, Cellular Pathology as Based Upon Physiological and Pathological Histology. Twenty Lectures Delivered in the Pathological Institute of Berlin during the months of February, March and April 1858., F. Chance trans. (Ed.), John, Churchill, London, 1860, pp. 196–219.
[15] R. Virchow, Gesammelte Abhandlungen zur Wissenschaflichen Medicin. Zweite Unveränderte Ausgabe. Hamm G. Grote'sche Buchandlung (C. Müller), 1862.
[16] Fresquet JL. Armand Trousseau (1801-1867). Historia de la medicina.org. in: Epónmos y biografias médicas, http://www.historiadelamedicina.org/trousseau.html 2008 (accessed 2 March 2019).
[17] A. Trosseau, H. Belloc, Traité Pratique de la Phthisie Laryngée, & c. A practical treatise on phthisis laryngeal, chronic laryngitis, and diseases of the voice, Edinb. M. & S. J. 48 (1837) 460-482.
[18] A. Trousseau, H. Belloc, A Practical Treatise of Laryngeal Phthisis, Chronic Laryngitis, and Disease of the Voice. Prize Essay of the Royal Academy. Translated by JA Warder, Published by A Waldie, Philadelphia, 1839.
[19] V.C. Medvei, The History of Clinical Endocrinology: A Comprehensive Account of Endocrinology From Earliest Times to the Present Day, The Parthenon Publishing Group, UK and New York, 1993.
[20] M.D. Moore, T.J. Fahey, Armand Trosseau 1801–1867. in: Pasleka JL, Lee JA (Eds.), Surgical Endocrinopathes: Clinical Management and the Founding Figures, 2015, pp. 177–179. doi: doi:1-/1007/978-3-319-13662-2_29.
[21] M.A. Samuels, M.E. King, U. Balis, Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 31-2002. A 61-year-old man with headache and multiple infarcts, N. Engl. J. Med. 347 (2002) 1187–1194. doi:10/1056/NEJMcpc020117.
[22] N. Callander, S.I. Rapaport, Trosseau's syndrome, West. J. Med. 158 (1993) 364–371.
[23] A. Trousseau, Lecture on clinical medicine: delivered at the Hotel-Dieu, Paris, Volume 5. Phlegmasia Alba Dolens (lecture XCV), in: Cormack JR, Bazire PV trans. (Eds.), Lindsay & Blakiston, Phladelphia, 1873, pp. 281-332.
[24] R.B. Birrer, C.B. Birrer, Medical Diagnostic Signs, Charles C Thomas Publisher, Illinois, 1982.
[25] R.A. Mahler, Thrombose, Lungenembolie und plötzlicher Tod, in: G. Leopold (Ed.) Geburtshülfe und Gynäkologie. Arbeiten aus der Dresdener Frauenklinik in Dresden. Hirzel. (VIII, 391 S. Lex. 8) e/24. Leipzig, 1895 pp. 72–120. [Original reference not accessible].
[26] C.G. Cumston, Pulmonary Embolus Following Operative Interference, Boston M. & S. J. 158 (1908) 284–287.
[27] E. Zurhelle, Thrombose und Embolie nach gynäkologischen Operationen, Archiv. für Gynäkologie. 84 (1908) 443–512.
[28] E.P. Davis, Obstetrics Thrombosis and Embolism in the Puerperal Period. Junge (Archiv f. Gynäkologie, 1912, Band xcvi, Heft 2), Am. J.M Sci. 147 (1914) 772-774.
[29] F. Gundlach, Catalogus Professorum Academiae Marburgensis (1527–1910), Elwert, Marburg, 1972.
[30] A. Rieländer, Über Venethrombosen im Wochenbett [about venous thrombosis in the puerperium], Monatschr. f. Geburtah. u. Gynaek, Berl. 24 (1906) 154–169.
[31] W. Pschyrembel, Pschyrembel Klinisches Wörterbuch mit klinischen Syndromen und Nomina Anatomica [Pschyrembel's Clinical Dictionary with Clinical Syndromes and Nomina Anatomica], Walter de Gruyter, Berlin, 1986.
[32] H. Michaelis, Prodromalerscheinungen der puerperalen und postoperativen Thrombose und Embolie [Prodromal signs of puerperal and postoperative thrombosis and embolism], München. med. Wohnschr. 58 (1911) 73-76.
[33] Anonymous, Current medical literature: premonitory signs of thrombosis, JAMA 56 (1911) 549.
[34] Joseph Ducing. Wikipédia L'éncylopédie libre. https://fr.wikipedia.org/wiki/Joseph_Ducuing 2016 (accessed 2 May 2018).
[35] Arlet J. Éloge de Monsieur le Doyen Lazortes. http://academie-sciences-lettres-toulouse.fr/wp-content/uploads/2017/10/2015-1-Eloge-Lazorthes.pdf (accessed 2 May 2018).
[36] R. Boure, Inventaire des hommages rendus aux chercheurs Toulousains par leurs pairs (années 1880–2010): attributions de noms, publications dédiées et galeries de portraits, Mars (2017).
[37] J. Ducuing, Phlébites Thromboses et Embolies Post-Opératoires, Masson et Cie, Paris, 1929.
[38] F.A. Schneider, I.R. Siska, J.A. Avram, Clinical Physiology of the Venous System, Kluwer Academic Publishers, New York, 2003.
[39] R. Janzen, Pain Analysis: A Guide to Diagnosis, Williams & Wilkins, Baltimore, 1970.
[40] H. Nüllen, T. Noppeney, C. Diehm, Venöse Thromboembolien [Venous Thromboembolism], Springer-Verlag, Berlin, 2014.
[41] E.B. Ringelstein, Prävalenz der tiefen Beinvenenthrombose bei Patienten mit wahrscheinlicher paradoxer Embolie im Vergleich zu Patienten mit anderer Insultätiologie [Prevalence of deep vein thrombosis in patients with probable paradoxical embolism compared to patients with other insultiology.]. Inaugural-dissertation zur erlangung des Doctor Medicinae der Medizinischen Fakultät der Westfälischen Wilhelms-Universität Münster vorgelegt von Schomacher, Guido aus Beckum 2007. https://kipdf.com/inaugural-dissertation_5ad8729f7f8b9a688b8b45bc.html 2007, (accessed 12 May 2018).
[42] M.M. Kochen, Management der tiefen Beinvenenthrombose: Veränderungen in Diagnostik und Therapie im Zeitraum von 1990 bis 2003 [Management of deep vein thrombosis: changes in diagnosis and therapy in the period from 1990 to 2003]. Inaugural-Dissertation zur Erlangung des Doktorgrades der Medizinischen Fakultät der Georg-August-Universität zu Göttingen vorgelegt von Felix K. Schlehahn Aus Leipzig Göttingen https://ediss.uni-goettingen.de/bitstream/handle/11858/00-1735-0000-0006-AF41-C/schlehahn.pdf?sequence=1 2007, (accessed 12 May 2018).
[43] J.I.F. Solares, J.L.P Garcia, C.M. Hernandez, M.F. Vargas, Diagnóstico de la trombosis venosa profunda y de la tromboembolia pulmonar [Diagnosis of deep vein thrombosis and pulmonary thromboembolism]. Documento de Consenso CEFyL Semergen. Recomedaciones sobre Profilxis, Diagnóstico y Tratamiento de la Enfermedad Tromboembólica Venosa en Atención Primaria. http://www.capitulodeflebologia.org/download/Mailings/Consenso-ETEV-en-Atencion-Primaria.pdf (accessed 18 May 2018).
[44] Segno di Laurel, http://happyslide.net/doc/323165/segno-di-laurel , Accessed

date: 20 May 2018.
[45] H. Friess, I.E. Demir, G.O. Ceyhan, Vereinigung der Bayerischen Chirurgen (1911-2011): Zur 100. Jubiläums-Tagung der Vereinigung [Association of Bavarian Surgeons (1911-2011): For the 100th Anniversary Conference of the Association]. München, http://www.vereinigung-bayerischer-chirurgen.de/content/files/100-jahre-vbc-buch.pdf 2011 (accessed 10 May 2018).
[46] K. Denecke, Der Plantarschmerz als Frühsymptom einer beginnenden Thrombose der unteren Extremität [Plantar pain as an early symptom of incipient lower extremity thrombosis], München. med. Wohnschr. 76 (1929) 1912–1913.
[47] John Olow, https://sv.wikipedia.org/wiki/John_Olow, (2018) , Accessed date: 15 March 2019.
[48] J. Olow, Sur un détail concernant le diagnostic de la thrombose crurale [On a detail concerning the diagnosis of crural thrombosis], Acta Obstet. Gyn. Scan. 10 (1930) 159–162.
[49] B. Haeseker, Dr. J.F.S. Esser and his Influence on the Development of Plastic and Reconstructive Surgery, Erasmus Universiteit, Rotterdam, 1983.
[50] F.H. Garrison, An Introduction to the History of Medicine, 3rd edition, W.B. Saunders Company, Philadelphia and London, 1921.
[51] Antoni Tomasz Jurasz. i jego szkoła chirurgiczna. Uniwersytet Medyczny im. Karola Marcinkowskiego w Poznaniu. Fakty UMP. Dwumiesięcznik informacyjny Nr 4/2016: 6–8.
[52] S.V. Linetskiy, H. Hubálková, H. Staňková, R. Šmucler, J. Mazánek, Ozone and its usage in general medicine and dentistry, Prague Med. Rep. 109 (2008) 5–13.
[53] Health Technology Assessment Unit a Medical Development Division Ministry of Health. Ozone Therapy. Health Technology Assessment Unit Medical Development Division Ministry of Health MOH/PAK/110.06 (TR), (2005) http://www.moh.gov.my/update2017/1009.pdf (accessed 30 April 2018).
[54] S. Pardeep, S. Kirtika, S. Neha, D. Nidhi, Review of ozone and its role in prosthodontics, Sch. J. Dent. Sci. 4 (2017) 226–232.
[55] S. Pagadala, D.C. Tadikonda, Ozone therapy a frontier in periodontics: a review, Int. J. Adv. Res. 3 (2015) 652–657.
[56] W. Drumi, C. Drumi, Emerich Ullmann (1861-1937): not only a pioneer of kidney transplantation, J. Nephrol. 17 (2004) 461–466.
[57] Wicht J.H. Renal transplant survey: how standardized is a standard kidney transplant. Thesis: Master of Medicine (MMed) in Surgery, University of Cape Town. https://open.uct.ac.za/bitstream/handle/11427/24507/thesis_hsf_2017_wicht_jonathan_h.pdf?sequence=1, 2016 (accessed 4 March 2019).
[58] D. Hamilton, A History of Organ Transplantation: Ancient Legends to Modern Practice, University of Pittsburgh Press, Pittsburgh, 2012.
[59] O.S. Schindler, Surgery for anterior cruciate ligament deficiency: a historical perspective, Knee Surg. Sports Traumatol. Arthosc. 20 (2011) 5–47, https://doi.org/10.1007/s00167-011-1756-x.
[60] V. Sanchis-Alfonso, J.C. Monllau, The ACL-Deficient Knee. A Problem Solving Approach, Springer-Verlag, London, 2013.
[61] E. Payr, Der heutige Stand der Gelenkchirurgie Verhandlungen der Deutschen Gesellschaft für Chirurgie [Current state of joint surgery sessions of the German Society of Surgery] 21st Congress, Arch. f. klin. Chir., Berl. 148 (1927) 404-521.
[62] A.J. Milojević, B.S. Radojčić, I.D. Meljnikov, Hydrocephalus-history of surgical treatment over the centuries, Istorija medicine 7 (2012) 119–125.
[63] I.S. Sivayoham, Camero-venous shunt for secondary glaucoma following orbital venous obstruction, Br. J. Opthalmol. 52 (1968) 843–845.
[64] Erwin Payr, Wikipedia: the free encyclopedia, https://en.wikipedia.org/wiki/Erwin_Payr, (2017) , Accessed date: 10 April 2018.
[65] V.E. Crolley, N. Gibbins, One hundred years of external approach medialisation thyroplasty, J. Laryngol. Otol. 131 (2017) 202–208.
[66] Spiegel HU. Aus dem Universitätsklinikum Münster Klinik und Poliklinik für Allgemeine Chirurgie [From the University Hospital Münster Clinic and polyclinic for general surgery]. Direktor: Univ. Prof. Dr. Norbert Senninger Abteilung Chirurgische Forschung Leiter: Prof. Dr. Dipl. Ing. H.U. Spiegel Die Entwicklung der „Chirurgischen Forschung“ in Deutschland. Inaugural-Dissertation zur Erlangung des doctor medicinae dentium der Medizinischen Fakultät der Westfälischen Wilhelms- Universität Münster vorgelegt von Neumann, Michaela, Brigitte aus Berlin, https://d-nb.info/991641264/34 2006 (accessed 8 May 2018).
[67] Erwin Payr. Austria-Forum https://austria-forum.org/af/AustriaWiki/Erwin_Payr (accessed 8 May 2018).
[68] G. Tschmarke, Erfahrung über den Fußsohlendruckschmerz als Frühsymptom der Thrombose [Experience on foot sore pressure pain as an early symptom of thrombosis], München. med. Wohnschr. 11 (1931) 2135.
[69] G. Tschmarke, Erfahrung über den Fußsohlendruckschmerz als Frühsymptom der Thrombose, Chirurg 3 (1931) 924–927.
[70] E. Payr, Gedanken und Beobachtungen über die Thrombo-Emboliefrage [Thoughts and observations about the thromboembolic issue], Centrabl. f. Chir., Leips., 57 (1930) 961-978.
[71] O. Meyer, Phlebitis: The Hidden Cause of Most Leg and Foot Ailments, Savoy Book Publishers, New York, 1940.
[72] O. Meyer, Symptomenkomplex und Therapie des Ulcus phlebiticum [Complex symptoms and therapy of the ulcer phlebiticum], München. med. Wohnschr. 79 (1932) 551.
[73] O. Petter, K. Holzegel, Zur geschichte der deutschen phlebologie, in: D. Kopielski (Ed.), Zur geschichte der deutschen phlebologie, Torgau Satz/Layout Druck, Germany, 2005, p. 22.
[74] E. Krieg, Zum klinischen Bild der infektiösen Fernthrombose [To the clinical picture of infectious post-operative thrombosis], München. med. Wohnschr. 82 (1935) 776–778.
[75] R.D. Strous, M.C. Edelman, Eponyms and the Nazi era: time to remember and time for change, Isr. Med. Assoc. J. 9 (2007) 207–214.
[76] Neumann Robert Dr. Startseite/1933-1945 Täter und Mitläufer/1933-1945 Biografien N. http://www.tenhumbergreinhard.de/1933-1945-taeter-und-mitlaeufer/1933-1945-biografien-n/neumann-robert-dr.html (accessed 10 April 2018).
[77] S. Hildebrandt, The Anatomy of Murder: Ethical Transgressions and Anatomical Science During the Third Reich, Berghahn Publications, New York, 2016.
[78] Robert Neumann (Pathologie). Wikipedia - Die freie Enzyklopädie. https://de.wikipedia.org/wiki/Robert_Neumann_(Pathologe) (accessed 7 May 2018).
[79] R. Neumann, Ursprungszentren und Entwicklungsformen der Bein-Thrombose [Centers of origin and developmental forms of leg thrombosis], Arch f. path. Anat. etc, Berl, 301 (1938) 708-735.
[80] Z.V. Maizlin, N. Westermark, Sans Atout and Olympic medal, Wonders of Radiology: Not Only for Wondering Radiologist, Create Space Publishing, 2010, pp. 138–141.
[81] Z.V. Maizlin, P.L. Cooperberg, J.J. Clement, P.M. Vos, C.L. Coblentz, People behind exclusive eponyms of radiologic signs (Part II), Can. Assoc. Radiol. J. 61 (2010) 44–53.
[82] Nils Westermark Sailor and Radiologist. https://upclosed.com/people/nils-westermark/ (accessed 2 February 2019).
[83] Nils Westermark. Archive.today. SR/Olympic Sports. Sports Reference LLC. http://archive.li/iBYNv, 2000-2015 (accessed 3 February 2019).
[84] N.J.H. Westermark, Roentgen Studies of the Lungs and Heart, University of Minnesota Press, Minnesota, 1948.
[85] N. Westermark, On the roentgen diagnosis of lung embolism, Acta Radiol. 19 (1938) 357–372.
[86] D.F. Worsley, A. Alavi, J.M. Aronchick, J.Y. Chen, R.H. Greenspan, C.E. Ravin, Chest radiographic findings in patients with acute pulmonary embolism: observations from the PIOPED Study, Radiology 189 (1993) 133–136.
[87] Robin Sturtevant Pilcher. Lives of the fellows. Royal College of Physicians. http://munksroll.rcplondon.ac.uk/Biography/Details/3567 (accessed 15 May 2018).
[88] R. Pilcher, Postoperative thrombosis and embolism, Lancet 234 (6055) (1939) 629–630.
[89] Z.V. Maizlin, P.L. Cooperberg, J.J. Clement, P.M. Vos, C.L. Coblentz, People behind exclusive eponyms of radiologic signs (Part I), Can. Assoc. Radiol. J. 60 (2009) 201–212.
[90] R. Schatzki, J.R. Lingley, Aubrey O. Hampton, 1900-1955, Am. J. Roentgenol. Radium Therapy, Nucl. Med. 76 (1956);396.
[91] A.D. Hampton, B. Castleman, Correlation of post mortem chest teleroentgenograms with autopsy findings with special reference to pulmonary embolism and infarction, Am. J. Roentgenol. 43 (1940) 305–326.
[92] J.S.T. Yao, The Society of Vascular Surgery John Homans lectureship, J. Vasc. Surg. 23 (1996) 1077–1080.
[93] N.R. Barshes, M. Belkin, The Homans family in American surgery, Ann. Surg. 261 (2015) 416–420.
[94] D.C. Elkin, John Homans lecture, N. Engl. J. Med. 245 (1951) 997–1000.
[95] F.J. Veith, A look at the future of vascular surgery, J. Vasc. Surg. 64 (2016) 885–890.
[96] J. Homans, Exploration and division of the femoral and iliac veins in the treatment of the thrombophlebitis of the leg, N. Engl. J. Med. 224 (1941) 179–186.
[97] A.W. Allen, R. Liton, G.A. Donaldson, Thrombosis and embolism: review of 202 patients treated by femoral vein interruption, Ann. Surg. 118 (1943) 728–739.
[98] J. Homans, Diseases of the veins, N. Engl. J. Med. 231 (1944) 51–60.
[99] E.L. DeGowin, R.L. DeGowin, Bedside Diagnostic Examination, MacMillan Publishing Company, New York, 1981.
[100] A. Felder, Evaluation of the various clinical signs of thrombophlebitis and experience in therapy with anticoagulants, Surg Gynecol Obstet 88 (1949) 337–350.
[101] S. Anand, P. Wells, D. Hunt, P. Brill-Edwards, D. Cook, J.S. Ginsberg, Does this patient have deep vein thrombosis? JAMA 279 (1998) 1094–1099.
[102] H.M. Seidel, J.W. Ball, J.E. Dains, G.W. Benedict, Mosby Guide to the Physical Examination, Mosby, St. Louis, 1999.
[103] J.E. Dunphy, T.W. Botsford, Examination of the blood vessels and lymphatics, Physical Examination of the Surgical Patient and Introduction to Clinical Surgery, W.B. Saunders Company, Philadelphia, 1975, pp. 228–257.
[104] S.R. Kahn, The clinical diagnosis of deep venous thrombosis: integrating incidence, risk factors, and symptoms and signs, Arch. Intern. Med. 58 (1998) 2315–2323.
[105] J. McLachlin, T. Richards, J.C. Paterson, An evaluation of clinical signs in the diagnosis of venous thrombosis, Arch. Surg. 85 (1962) 738–744.
[106] F.L. Urbano, Homans' sign in the diagnosis of deep vein thrombosis, Hosp. Physician 37 (2001) 22–24.
[107] D.A. Sandler, Homans' sign and medical education, Lancet 326 (8664) (1985) 1130–1131, https://doi.org/10.1016/S0140-6736(85)903723-8.
[108] N. Shafter, S. Duboff, Physical signs in the early diagnosis of thrombophlebitis, Angiology 22 (1971) 18–30.
[109] B.A. Naib, N.A. Salih, Accuracy of color duplex imaging (CDI) in diagnosis deep vein thrombosis (DVT) versus clinical predication index, I.J.S.R. 2 (2013) 66–70.
[110] K. Haeger, Problems of acute deep venous thrombosis, the interpretation of signs and symptoms, Angiology 20 (1969) 219–223.
[111] E.W. Knox, Deep thrombophlebitis of the lower limbs, Ulster Med. J. 33 (1964) 28–35.
[112] B. Miles, Candles in Denmark, John Murray Publishers, London, 1958.
[113] H. Bisgaard, Ulcus und Eczema cruris, Phlebitidis sequelae usw: studien über die statischen Erkrankungen der unteren Extremitäten und ihre Behandlung, Kopenhagen (1941).
[114] A.M. Wiersema, The history of heparin. Part II: The early development and road to clinical use, Periprocedural Prophylactic Antithrombotics in Arterial Procedures: The Road to Consensus, GVO/Ponsen & Looijen, Netherlands, 1964, pp. 43–81.

[115] B. Eklöf, Presidential address: the dynamic approach to venous disease-following in the footsteps of Gunnar Bauer and Robert Kistner, J. Vasc. Surg. 42 (2005) 369–376.
[116] G. Bauer, Venous thrombosis, early diagnosis with the aid of phlebography and abortive treatment with heparin, Arch. Surg. 43 (1941) 462–472.
[117] GJ Curry, I Cohn, F.V.. Wade, Frederic Wolcott Bancroft, in: C.C. Thomas, (Eds.) Profile in Trauma: Illinois; 1969, pp. 15-17.
[118] N.Y. Anonymous, Med. J. 83 (1906) 1306.
[119] Catalogue of Officer and Graduates Columbia University from the Foundation of King's College in 1754. 16th edition., Columbia University, New York, 1916.
[120] Columbia University Bulletin of Information. Annual Reports of the President and Treasurer to the Trustees With Accompanying Documents for the Year Ending June 30, 1917.
[121] L.F. Peltier, Fractures: A History and Iconography of Their Treatment, Norman Publishing, San Francisco, 1990.
[122] Reprints from the Surgical Service New York City, Fifth Avenue Hospital, 1928.
[123] Frederic W. Bancroft, New York Physician 6 (1936) 13.
[124] Columbia-Presbyterian Medical Center, The center, Annu. Rep. 4 (1962) 155.
[125] F.W. Bancroft, Proximal ligation and thrombectomy for phlebothrombosis of the femoral and iliac veins, Ann. Surg. 121 (1945) 175–184.
[126] M. Fric, J. Pechán, M. Mikulecký, M. Valentín, I. Simko, Evaluation of clinical signs and symptoms in active deep venous thrombosis of the calf, Cor Vasa. 27 (1985) 346–352.
[127] W. Molloy, J.J. English, R. O'Dwyer, J. O'Connell, Clinical findings in the diagnosis of proximal deep vein thrombosis, Ir. Med. J. 75 (1982) 119–120.