International Journal of Cardiovascular Sciences. 2016;29(5):396-406

REVIEW ARTICLE

Cardiovascular Semiotics: The Personalities Behind the Eponyms

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Abstract

Since its inception, medicine has been based on observation of signs and specific findings in ill patients. Semiotics is, therefore, an ancient study. Cardiac semiology, although more recent, is more complex in its learning due to difficulties in the interpretation of auscultatory findings. Austin Flint, Rivero Carvallo, Antonio Valsalva, and Adolf Kussmaul are some of the many physicians who have dedicated themselves to the academic study of cardiac semiology and became eternalized in the medical field through eponyms in cardiology. A selection of the main and most iconic eponyms in cardiology is necessary to complement and highlight the importance of the knowledge of cardiovascular physical examination at the bedside and the interpretation of its findings, especially in an era in which medicine is involved with so many technological innovations in the imaging area. The aim of this review article is to address historical aspects of selected cardiologic eponyms and the importance of these eponyms in current medical practice, especially for those in training who want to deepen their study of cardiovascular semiotics.

Introduction

The cardiovascular physical examination is one of the most difficult skills acquired by physicians in their training. The increasing technological incorporation observed in life today is also present in medicine and its teaching. Currently, many teachers and students tend to neglect cardiac auscultation in favor of imaging examinations to evaluate the heart, such as echocardiography and magnetic resonance imaging. However, it is worth noting that, in many cases, the reality of medicine distances patients from access to cardiovascular imaging examinations, even in large centers.

The lack of competence for cardiac auscultation can be seen in several countries around the world. Mangione *et al.*[1] assessed the accuracy of auscultation performed by residents in internal medicine in the US, Canada, and England and found that the correct assessment of the cardiovascular condition was carried out in only 22%, 26%, and 20% of the cases, respectively.[1] National data are not available; however, we probably present similar rates of inability to perform an accurate physical examination.

Cardiac semiology has been developed over many decades. In fact, centuries have passed until we reached the current state of knowledge, and the modern understanding of circulatory abnormalities and their visible and noticeable clinical impact is a very important skill in cardiological practice. Technology should be used as an additional tool to the physical examination, as shown by our group of students when we use the digital stethoscope in our ward practices.[2]

The study and understanding of the main phenomena of clinical examination that receive their own nomenclature (eponyms) is a valuable opportunity to review the clinical examination and understand the evolutionary aspects of cardiology. The approach to the biography of important physicians who name these eponyms becomes, then, a

Keywords

Anamnesis; Physical Examination; Diagnostic Techniques, Cardiovascular; Eponyms.

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DOI: 10.5935/2359-4802.20160063

Manuscript received June 30, 2015; revised manuscript July 17, 2016; accepted September 30, 2016.

stimulus for the study and appreciation of cardiovascular semiotics. The knowledge of the personalities behind the eponyms instigates the physician and those in training and assists in the consolidation of knowledge. We chose some of the most important signs and maneuvers in cardiology to serve this purpose.

Austin Flint Murmur

Austin Flint was an American physician from the state of Massachusetts. The son of a father and grandfather, who were both physicians, his family started in medicine in 1638. He was a very influential physician dedicated to medical schools, and he innovated teaching methods to encourage his students to conduct clinical case studies. Extremely scholarly, he wrote more than 240 articles, including the initial description of cases of rubella in 1840 and a fever epidemic, later known as typhoid fever.[3] Furthermore, he published the book *A Treatise on the Principles and Practice of Medicine*, one of the most influential in the medical field at that time.

In 1859, Flint observed two cases of patients with aortic insufficiency who had a presystolic murmur similar to that present in mitral stenosis, despite an intact mitral apparatus at autopsy.[3] At that time, Austin Flint practiced medicine at the Charity Hospital. The finding was originally described as a murmur caused as a result of ventricular filling before atrial contraction, in which the mitral valve closes ahead of time; in these circumstances, there is a relative mitral stenosis, despite the absence of a valvular lesion.[3] The finding was then published in 1862 in *The American Journal of The Medical Sciences* through the article *On Cardiac Murmurs*.[3] Some years later, it was observed that the murmur could also be located in the middle systole or throughout systole and it, thus, received the name of the physician who initially notified it.[4]

Currently, the etiology of the Austin Flint murmur is still very controversial,[5] and several hypotheses have already been suggested to explain it, including a vibration of the anterior leaflet of the mitral valve due to aortic regurgitation, collision between the regurgitated blood flow and the mitral leaflet, vibration resulting from the regurgitant jet in the free wall of the left ventricle, and a reduction in the mitral valve orifice that changes the speed of blood flow from the left atrium.[6]

As the Austin Flint murmur is indistinguishable from the murmur caused by mitral stenosis, one study aimed to identify findings able to distinguish the murmur due to isolated severe aortic insufficiency from that present in patients with aortic insufficiency associated with mitral stenosis.[7] The patient that presents an Austin Flint murmur is usually male, with progressive symptoms of both dyspnea, as well as angina *pectoris* and sinus rhythm. The cardiac auscultation shows the second heart sound with a normal or slightly accentuated intensity, and a first muffled heart sound which tends to disappear. A systolic ejection murmur in the areas at the base of the heart, most often throughout the precordial, can be observed. Auscultation of the heart apex shows a systolic murmur, diastolic ventricular gallop, irradiated presystolic murmur of the aortic area, and diastolic rumbling often accentuated in middle systole.[7]

The sensitivity of this semiological finding varies widely according to the literature. For patients with severe aortic regurgitation, its sensitivity ranges from 57%[8] to 100%,[9] while in patients with a mild valvular pathology, sensitivity rates between 0% and 50% have been observed.[10] Quantifying the specificity of this finding is not necessary since this murmur is unique to aortic insufficiency.[11]

Corrigan Pulse

James Hope and Thomas Hodgkin were the first physicians to describe the signs of aortic insufficiency in the years 1826 and 1827, respectively.[12,13] Aortic insufficiency is a clinical syndrome in which the aortic valve lacks proper closure during cardiac diastole. Consequently, there is passage of blood from the aorta into the left ventricle causing an increase in the end-diastolic volume, which overloads volumetrically the heart. This syndrome has a pleiad of symptoms and signs, of which one of the best known is the Corrigan Pulse.

In 1832, the Irishman Dominic John Corrigan was responsible for describing in detail the outline of the carotid pulse in patients with aortic insufficiency and correlate it with the associated valvular pathology.[14] Corrigan's description was based only on observation of the arterial pulse. He noted that when patients with aortic insufficiency were undressed, his gaze was directed to the pulsation of the arteries in the head, neck, and upper extremities, which showed a very different pattern. Corrigan observed that during diastole, there was a rapid reduction in the diameter of the arteries. Whereas, when ventricular contraction occurred, the arterial diameter increased suddenly, making the pulsation quite visible. This was best observed in the upright position than in the horizontal one.

In relation to the arteries of the extremities, Corrigan did not observe differences in pulsation. However, he realized there was increased pulsation of the brachial and palmar arteries when the patient raised the upper limb perpendicular to the head.[15] In 1843, Thomas Watson described the palpatory characteristics of aortic insufficiency, calling them the water-hammer pulse; however, as Corrigan described the pulse based on visual aspects, we do not consider the signs to be synonymous, despite being used as such in clinical practice. Therefore, the water-hammer pulse, or Corrigan Pulse, is a fairly quick arterial pulse in which there is a rapid and elevated increase in amplitude during systole, followed by a rapid collapse during diastole (Figure 1).[16]

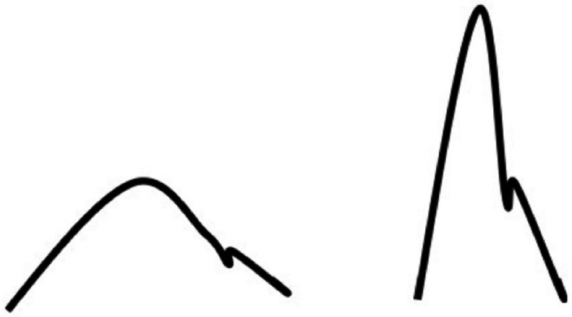

Figure 1
Illustrated representation of the outline of the carotid pulse. On the left, the outline of a normal pulse and, on the right, the outline of the Corrigan Pulse.

De Musset's Sign

Another well-known sign in patients with aortic insufficiency is the de Musset sign,[4] due to a movement of the patient's head in relation to the heartbeat. Just like other semiological findings, the de Musset sign reflects the increase in systolic pressure due to a larger end-diastolic volume.[17]

Unlike most eponyms in medicine, in which the semiological sign or pathology takes the name of the physician who reported the case, the de Musset sign receives the name of the patient in whom this sign was described. Alfred de Musset (1810 – 1857) was a French poet and playwright who had aortic insufficiency secondary to syphilitic aortitis. His brother, Paul de Musset, was who first described the sign bearing the name of his blood relative, when he wrote a biography on Alfred de Musset (*Biography of Alfred de Musset, 1864*). During a lunch, Paul de Musset noted that his brother moved his head rhythmically, involuntarily, and synchronously with the heartbeat. Such movement would disappear with a pressure applied on the neck in the region of the carotids.[18,19] It was only in 1900 that Andre Delpeuch proposed naming the sign with the name of the poet.[18]

The de Musset sign is nonspecific, demonstrating low sensitivity and specificity in the diagnosis of aortic insufficiency.[20]

Rivero-Carvallo Maneuver

José Manuel Rivero Carvallo was a prominent Mexican physician, responsible for creating the first residency in cardiology, the first cardiology journal, and the first specialty hospital in the country.[21] Carvallo was born in 1905 in Puebla, Mexico. He studied medicine in Puebla, and in 1928 continued his studies in Paris, where he completed his training in 1930. In 1932, he obtained his Doctoral Degree from the University of Sorbonne, with a thesis entitled *La préssion artérielle au tours de l'anesthésie et de certaines interventions chirurgicales* ("Variations in arterial pressure during anesthesia and certain surgical interventions").[22]

In 1946, Carvallo published an article entitled "*Signo para el diagnóstico de las insuficiencias tricuspídeas*" ("Sign for diagnosis of tricuspid insufficiencies") in the journal *Archivos del Instituto de Cardiologia de México*.[23] Rivero Carvallo employed a maneuver that consisted in asking the patient to perform deep breaths and to

maintain inspiratory apnea, testing this maneuver on four different groups of patients with (1) clinical evidence of tricuspid insufficiency, (2) clinical evidence of mitral lesion without tricuspid involvement or arrhythmias, (3) rheumatic tricuspid valve and arrhythmias, and (4) heart failure or arrhythmia with no rheumatic disease. The resulting sign produced by this maneuver was an increased intensity of systolic murmur in patients with tricuspid insufficiency, of maximum intensity in the tricuspid area and irradiation to the mesocardium and hepatic region. In addition to the increased intensity, Rivero Carvallo also described a change in the tone of the murmur that would become crude, like a "jet of steam", which became known as the Rivero-Carvallo maneuver.[22]

In one study with Dr. Isaac Costero, Rivero Carvallo found that the maneuver was confirmed after death in 10 out of 11 autopsies of patients with tricuspid insufficiency.[22] In another study, conducted by Dr. B. L. Fushdeler, the maneuver was positive in 75% of 300 studied cases of tricuspid insufficiency.[23]

In 1950, Rivero Carvallo described the use of his maneuver also for the diagnosis of tricuspid stenosis. In this case, an increase was observed in the intensity of the diastolic murmur and the opening snap.[24]

The pathophysiology of these phenomena can be explained by the fact that, during deep breathing, the intrathoracic pressure reduces, increasing blood return to the right side of the heart. Thus, an increase in the pressure, speed, and volume of blood through the tricuspid valve occurs, intensifying the sounds originating from the pathologies of this valve (Figure 2).[22]

In 1965, along with Helena Ramirez-Jaime, Carvallo observed that with the progression of the disease, the diastolic murmur could disappear in some patients due to right atrial dilation associated with the thinning of the atrial walls, which they called *atrium papyraceum* ("paper-thin atrium").[25]

Rivero Carvallo published several studies, such as those on rheumatic mediastinitis,[26] ectasia of the descending aorta,[27] apical impulse in tricuspid lesions,[28] atrial dilatation and dilation of the descending aorta,[28] double apical shock,[29] among others. Carvallo is considered one of the greatest masters in Mexican cardiology, and his maneuver is the most used and known Mexican eponym.

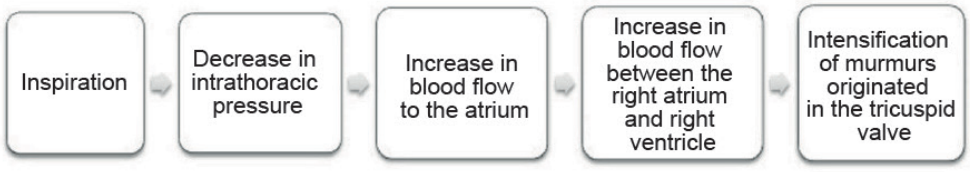

Figure 2
Schematic representation of the Rivero-Carvallo maneuver.

Kussmaul Sign

Adolf Kussmaul, born in 1822 in Germany, joined the renowned Heidelberg University to study medicine at the age of 18 years, where he won a gold medal for his final thesis on color changes in the fundus of the eye. After a brief stint working in the German army, he served as a physician in his country for a few years until he left medical practice, completing his degree as Doctor of Medicine in 1854. He then began his academic career teaching at the universities of Heidelberg, Freiburg, and Strasbourg.[30]

Kussmaul described numerous semiological phenomena of significant value in medical practice. He was the first to describe pericarditis nodosa (currently known as polyarthritis) and progressive bulbar palsy, to diagnose mesenteric embolism, to perform esophagoscopy and gastroscopy, in addition to being a pioneer in performing thoracentesis.[30] In addition to the Kussmaul sign, he described the paradoxical pulse in constrictive pericarditis and the Kussmaul breathing.[31]

In 1873, Kussmaul published *Ueber Schwielge Mediastino-pericarditis und den Paradoxen Puls* ("Concerning Callous Mediastinopericarditis and the Paradoxical Pulse") with the following description of a case report: "The jugular vein becomes considerably swollen" and "with each inspiration, a slight increase in its content

can be noticed." The sign that received the name of Kussmaul was then described for the first time, originally discovered by Greiginger in 1854, whom Kussmaul duly gave credit to in his publication.[31] This phenomenon is the visualization of the paradoxical increase in jugular venous pulse during inspiration, contrary to the normal physiological event, in which the jugular venous pressure falls with breathing.[32,33] Although it was originally described in constrictive pericarditis, the Kussmaul sign may be present in right ventricular failure, right ventricular infarction, tricuspid stenosis, massive pulmonary embolism and cardiomyopathic restriction.[32,34]

The pathophysiology of this phenomenon can be explained by an increase in right atrial pressure during inspiration, due to a restriction to right ventricular filling. In the case of cardiac tamponade, the Kussmaul sign cannot be visualized (Figure 3).[34]

At 66 years of age, he wrote his autobiography *Memories of the Youth of an Old Doctor*, which has been described as one of the most fascinating medical autobiographies ever written.[35]

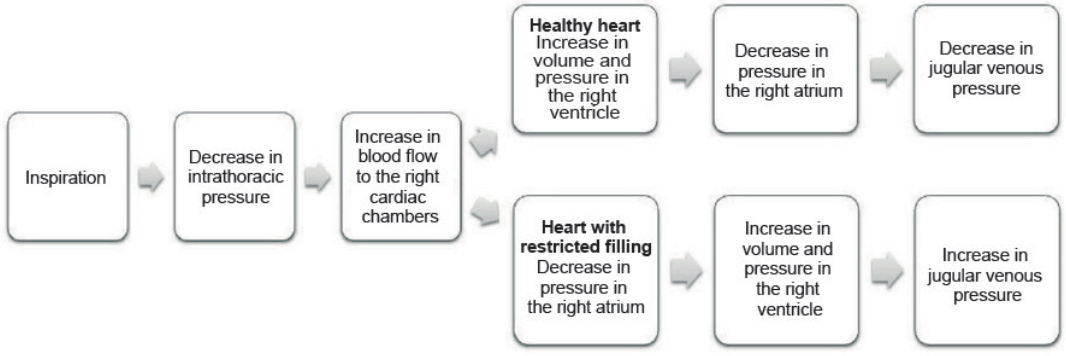

Figure 3

Schematic representation of the Kussmaul sign.

Graham-Steell Murmur

Graham Steell was a Scottish cardiologist in love with teaching at the bedside and with a special mastery in correlating physiology with clinical practice. Several years after graduating, while teaching in Berlin, London, and Manchester, he became known as the most important name in cardiology in northern England.[36] Gentle and kind with patients, he was reserved and extremely shy.[37] He authored several monographs related to physical examination for medical students and wrote a book, *Text Book on Diseases of the Heart*, which was highly commended. He liked to point out clinical symptoms and discuss their importance in the clinical condition of the patient, but he always stressed that the diagnosis could not be trusted in a single observed sign.[36] In contrast, his lectures were not much in demand, because his teaching method consisted of reading notes, with his head down and struggling to read his own writing, skipping entire passages.[38]

In 1888, Graham Steell described an early diastolic soft murmur along the left sternal border, related to increased pulmonary blood pressure (BP) secondary to mitral stenosis in patients without clinical signs of aortic insufficiency, in other words, pointing to the condition of pulmonary insufficiency.[39] His description was as follows: "Sometimes, in cases of mitral obstruction, it is auscultated in the pulmonary area (left sternal extremity in the third intercostal space) and up to one or two inches below, and rarely in the most distal part of the sternum, a soft diastolic murmur immediately after a second hyperphonetic heart sound, while the usual signs of aortic insufficiency verified by the pulse, *etc.*, are absent. The greatest intensity of the murmur is auscultated in the third and fourth left intercostal spaces at the sternal border. When the second heart sound is split, the murmur begins after the second component... the murmur of increased pulmonary artery pressure is not indicative of mitral stenosis, although it is most commonly found as a consequence of this lesion. Any obstruction of the continuous pulmonary circulation can produce it."[39]

The Graham-Steell murmur, as initially described, may not be an isolated finding and is associated with

additional findings of pulmonary arterial hypertension and right ventricular hypertrophy, such as a hyperphonetic second heart sound, increased middle arc on chest X-ray, electrocardiogram indicating overload of the right chambers, and absence of clinical signs of aortic insufficiency.[40]

An interesting twist, in relation to the naming of the eponym, was published in 1991 by Fraser *et al.*[37], who raised the hypothesis that there were descriptions previous to the one by the physician Graham Steell in a case reported by James Hope, John Hunter, and Dyce Duckworth. The article suggests that the eponym was given randomly, due to the fact that Graham Steell described it in a Medical Practice Treaty in 1898 and, therefore, it would not mean that he had named the murmur after himself, but that the readers had done that.[37] One of the concerns most expressed by Graham Steell, dated in 1900, was the fear that technology might alienate the physician from the contact with the patient.[41]

Valsalva Maneuver

Antonio Maria Valsalva was born in Italy in 1666.[42,43] Prior to studying medicine at the University of Bologna, he studied mathematics and philosophy. In 1687, he obtained the acceptance of his medical registration from that university to practice medicine and surgery. During medical school, he was a mentee of Marcello Malpighi, well-known physician and biologist.[42] In 1705, he became a professor of anatomy at the university where he had studied.[43]

Despite his great contribution to cardiology, this was not the only area in medicine to which Antonio Valsalva dedicated. In 1704, he published a treatise on the anatomy of the human ear entitled *De aure humana tractatus*, which became a reference for decades. He also described the aortic sinus that was named in his honor.[42,43] In that same year of 1704, Valsalva described that, after a normal inspiration, forcing an expiration with the nose and mouth closed would transmit increased pressure to the tympanic membrane that would assist in the expulsion of foreign bodies and pus from the middle ear.[44] Over the years, the physiological process of the maneuver has been studied by other physicians, and its use has expanded.

Nowadays, the Valsalva maneuver is described as an expiration with the glottis closed after a forced inspiration for at least 10 seconds. In order to assess the heart sounds, the inspiration should not reach the maximum, as it would alienate the thoracic wall of the heart and may mask some findings from the auscultation of the heart.[45] The physician should assess the patient's BP and, subsequently, inflate the cuff to 15 mmHg above the systolic BP. With the diaphragm of the stethoscope positioned in the topography of the patient's brachial artery, the examiner asks the patient to start expiration with occlusion of the airways. The examiner must be aware of changes in the BP.[43]

The maneuver consists of four stages. In phase I, the beginning of expiration with an obstructed airway generates an increase in intrathoracic pressure (ITP), which culminates in an immediate increase in stroke volume (SV) and compression of the aorta, leading to an increase in BP. During phase II, the persistence of respiratory effort will generate a decrease in venous return due to maintaining a high ITP, consequently leading to SV and BP fall. Phase III begins at the moment the expiratory effort ceases, decreasing the ITP and reducing the BP. Whereas, in phase IV, there is an activation of the sympathetic nervous system that will result in an increase in cardiac output and peripheral vasoconstriction, culminating in an elevation of the BP ("overshoot") (Figure 4).[43,46]

There are two changes that may arise in the maneuver that are compatible with heart failure and have high sensitivity and specificity:[47] the "square-wave" response, which may appear in patients with left ventricular dysfunction and is characterized by maintenance of the BP in phase II, and the absence of "overshoot" in phase IV.[43,45]

The great utility of the Valsalva maneuver in cardiovascular semiology is in the distinction of heart murmurs. Those originating in the right heart chambers can be audible early in phase IV, whereas those originating from the left chamber can be audible later in the same phase. In mitral and aortic stenoses, constrictive pericarditis, and interatrial communication, there may be a "square-root" response or an absence of "overshoot" in phase IV.[43,45] In mitral valve prolapse, there is anticipation of the click and a more evident and prolonged murmur.[47] Regarding hypertrophic cardiomyopathy murmur, there is an increase in intensity in phase II.[48] To distinguish the systolic murmur of this pathology from others, the maneuver has a sensitivity of 65% and specificity of 96%.[49] Cruveilhier-Baumgarten's periumbilical venous hum, present in cirrhotic patients, may also increase.[50]

An important function of the maneuver is the ability to interrupt an episode of supraventricular paroxysmal

tachycardia or ventricular tachycardia,[51,52] and help detect the long QT syndrome.[53] Another consequence may be the induction of a rate-dependent right bundle-branch block in phases II and III.[54] Finally, the Valsalva maneuver promotes an increase in clotting factor VIII in individuals with Von Willebrand's disease.[55]

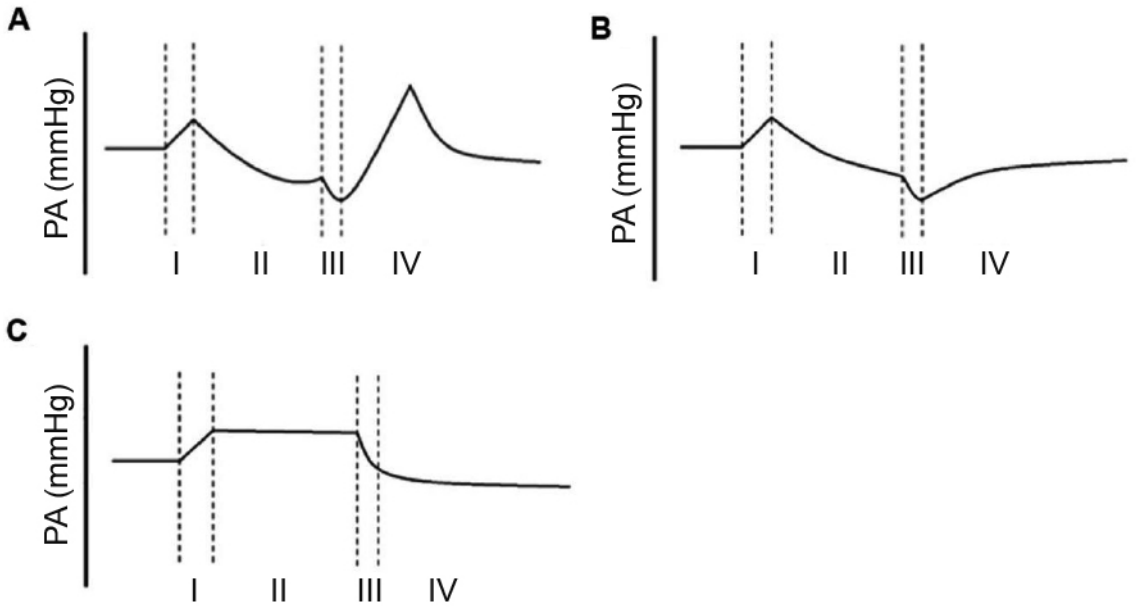

Figure 4

Variation in blood pressure (BP) during the Valsalva maneuver. During phase I, BP increases due to increased stroke volume (SV) and compression of the aorta. Phase II is characterized by a decrease in BP due to decreased venous return, resulting in a reduction of the SV. The end of the respiratory effort in phase III generates a decrease in intrathoracic pressure (ITP), leading to decreased BP. Finally, the BP is increased ("overshoot"), due to activation of the sympathetic nervous system, which causes peripheral vasoconstriction and increased cardiac output. (A) Sinusoidal response in a healthy patient. (B) Absence of "overshoot" in phase IV. (C) Square-wave response.

Janeway Lesions

Edward Gamaliel Janeway was born in New Jersey in 1841 and studied at the College of Physicians and Surgeons in New York. His first year in medical school was tumultuous because he disliked the detachment of theoretical studies from medical practice. In 1866, he began his medical practice at the Bellevue Hospital, initially as a pathologist and later as a professor and successor to the renowned physician Austin Flint, after his death in 1886.[56]

Janeway was an advocate of autopsies, and his passion was to correlate the pathological anatomy with clinical practice.[57] He was a very influential pathologist and researcher, mainly in the area of cardiovascular sciences, with articles on pulmonary gangrene, aortic aneurysm, and portal vein thrombosis.[56]

After consolidating his knowledge in pathology, he began to expand his interest and performance in clinical medicine, becoming one of the physicians responsible for creating the specialty of internal medicine in the United States.[56] In 1886, he joined William Osler, Francis Delafield, William Pepper, and other renowned physicians at that time, to found the American Medical Association.[58]

In 1899, he published a scientific article on heart diseases that included the description of lesions found in patients with infectious endocarditis: numerous small and slightly nodular palmoplantar hemorrhagic lesions.[59]

Janeway worked in several hospitals and was recognized for his brilliant reasoning and diagnostic power, acting as a consultant to physicians and hospitals.[56] He lived surrounded by students, one of

them, Emanuel Libman, who, when talking about endocarditis in a publication about blood cultures in 1906, cited that hemorrhagic and conjunctival petechiae and "slightly nodular small hemorrhages on the palms of the hands and soles of the feet" had a high degree of connection to endocarditis. Also on this point, he called said finding "Janeway lesions", with this being the first time that the eponym was used.[60]

The pathophysiological mechanism associated with Janeway lesions is related to septic microemboli of bacteria potentially capable of causing infection and that generate microabscesses in the dermis and thrombosis of small vessels.[61-63]

Gallavardin Phenomenon

Son of a homeopathic physician, Louis Gallavardin was born in the French city of Lyon in 1875.[64] He was a renowned French physician known for his impeccable ability of observation at the bedside. He carried out his professional training at the Lyon School of Medicine and became a physician in 1902.[65] Due to a legislative issue, he was forced to abandon his post at the hospital, but continued deepening his knowledge with research work.[64,66]

Between 1898 and 1945, Gallavardin was the author of 360 publications in various areas of cardiology, including cardiac arrhythmias, angina *pectoris*, myocardial infarction, and stenosis of the left heart valves, in addition to writing a treatise on diastolic hypertension.[64] As regards mitral pathology, the researcher described the clinical condition of pulmonary edema secondary to a reduction in the diameter of the valve orifice. Gallavardin was interested in aortic stenosis and was the first to emphasize the frequency of the non-rheumatic type of the disease, in addition to speaking about the frequent occurrence of effort syncope in this pathology.[65]

In the 1920s, the researcher described an auscultation finding in patients with aortic stenosis, which later became known as the Gallavardin Phenomenon.[67] This semiological finding consists in the transformation of a rough murmur originally present in the aortic area into an acute murmur with a musical pitch audible in the cardiac apex. This change occurs because of the musical components of the murmur being preferentially transmitted from the original area to the cardiac apex through solid tissues.[66] Thus, there would be two forms of irradiation from the murmur produced by the aortic stenosis: the rough noise originated by the valvular leaflets could transmit the sound to the blood vessels of the neck, and the mechanism described by the French physician.[68]

In the year 1925, the phenomenon addressed was published in volume 135 of the *Journal de Médecine* in Lyon in the document *Le souffle de rétrécissement aortique peut changer de timbre et devenir musical dans sa propagation apexienne*.[64] The literal translation of the publication's title is "The aortic stenosis murmur can change the pitch and become musical due to its propagation to the apex."

For many years, the mechanism of the systolic murmur with an audible musical pitch in the mitral area described by Gallavardin was widely accepted.[68] However, an article published in 1974 challenged the description of the researcher and assigned the noise in question to the association of an aortic stenosis with a mitral insufficiency secondary to a papillary muscle dysfunction.[64,68]

Beck's Triad

Richard Lower, an English physician, carried out in 1699 the first pathological clinical description of the physiology of cardiac tamponade. According to Lower, the amount of liquid present in the pericardial sac is sufficient to act as a lubricant and facilitate the movement of the heart. When the accumulation of a large amount of fluid in the pericardial sac occurs, there is compression of the walls in the heart, preventing sufficient ventricular dilatation to receive blood. Consequently, there is a reduction in the amplitude of the arterial pulse, which may cause, as the condition progresses, syncope and death. It was in the nineteenth century that this condition became known as "cardiac tamponade", its main clinical features were described, and the first attempt at treatment was performed.[69]

Cardiac tamponade presents several signs and symptoms, such as tachypnea and dyspnea at intense effort, which progress to dyspnea at rest. Furthermore, all patients present tachycardia,[70] paradoxical pulse, hepatomegaly, and Beck's Triad.

In 1935, Beck's Triad was described by Claude Schaeffer Beck, who was born in 1894, in Shamokin, Pennsylvania.[71] In 1917, Beck joined the John Hopkins School of Medicine. In 1924, he went to Cleveland[72] where he did his medical residency and specialized in cardiovascular surgery. Throughout his life, Beck dedicated much of his time to scientific research, especially those related to problems occurring during heart surgery. After years of research

and failed attempts,[72] Claude Beck performed in 1947 the first intraoperative defibrillation that led to resuscitation of a patient who presented ventricular fibrillation as the cardiorespiratory arrest rhythm.[73] In 1952, he received the title of professor of cardiovascular surgery, the first in the United States. At the age of 77 years, Beck died due to a stroke.[72]

Claude Beck understood the signs of acute cardiac tamponade, relating them to the physiological mechanisms of the condition, and composed the triad with (1) arterial hypotension, (2) jugular turgescence, and (3) muffled heart sounds.[74]

When a rapid accumulation of fluid occurs within the pericardial sac, there is an increase in pressure with compression of the heart and the part of the vena cava circumscribed by the pericardium. Consequently, blood is unable to flow from the systemic venous system into the heart chamber, which generates increasing central venous pressure and jugular turgescence. Due to the reduction in ventricular filling, hypotension occurs. However, due to the efficiency of the vasomotor center, the BP only reaches very low rates in very intense and acute cases. The muffled heart sounds are due to the increased distance between the heart and the thoracic wall.[75]

The famous Triad is considered pathognomonic of cardiac tamponade, but is only found in 10% to 40% of the cases.[76] Thus, it exhibits low sensitivity. Therefore, its absence should not exclude the diagnosis of cardiac tamponade.[77]

Currently, even in the absence of Beck's Triad, we are able to detect cardiac tamponade through complementary tests, and a patient suspected of having cardiac tamponade should be referred to transthoracic echocardiography for investigation.[78]

Conclusion

Knowledge of the origin of the main eponyms used in cardiovascular semiology enables a larger understanding of the history and development of cardiology for working professionals and medical students who are sometimes faced with names without proper knowledge of the framework during their training and practice of clinical examination. The deepening of this understanding facilitates learning and memorization of their meanings. Moreover, it allows a review of the clinical examination and understanding of how it evolved.

The eponyms addressed here show that the patient's anamnesis, ectoscopy, and physical examination should be the foundations of cardiovascular diagnosis, and should receive increased attention from physicians undergoing training. Through them, it is possible to diagnose cardiovascular diseases without the use of imaging tests, which are intended to supplement the clinical findings.

Author contributions

Conception and design of the research: Almeida RGP, Macaciel JS, Santos EAR, Garcia TCC, Hashimoto AM, Mesquita CT. Acquisition of data: Almeida RGP, Macaciel JS, Santos EAR, Garcia TCC, Hashimoto AM, Mesquita CT. Analysis and interpretation of the data: Almeida RGP, Mesquita CT. Statistical analysis: Almeida RGP. Writing of the manuscript: Almeida RGP, Macaciel JS, Santos EAR, Garcia TCC, Hashimoto AM. Critical revision of the manuscript for intellectual content: Almeida RGP, Macaciel JS, Santos EAR, Garcia TCC, Hashimoto AM, Mesquita CT.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

his study was partially funded by Pró-Reitoria de Gradução da Universidade Federal Fluminense.

Study Association

This study is not associated with any thesis or dissertation work.

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