



Physical Signs of Inspection and Medical Eponyms of Pericarditis Part II: 1864 to 1895

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ABSTRACT

During the mid- to late eighteenth century, physicians continued to make significant contributions describing their observations identified on physical examination in patients diagnosed with pericardial effusion or adherent pericardium. These diagnostic findings were eponymously named as signs in recognition of and to honor the contribution of physicians. The signs involve observation of the abdominal and chest wall during respiration and cardiac contraction, as well as changes occurring in the jugular veins during the cardiac cycle. These signs assisted physicians to further confirm the diagnosis and explain the pathogenesis of the underlying disease at a time where there were no imaging tests available. Observation of the height of the jugular venous wave and movements of the chest and abdomen wall during the cardiac and respiratory cycles provided physicians during this time period additional methods to detect pericardial effusion or adhesive pericarditis and mediastinitis. These findings depicted as sign of medical eponyms further enhance our understanding of the pathophysiological mechanism of disease. The absence of studies on these signs leads to a lack of insight about their accuracy and usefulness in modern-day clinical practice.

Keywords: Pericarditis, physical examination, eponyms, history of medicine

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INTRODUCTION

Inspection is the initial step performed during the physical examination. Information obtained through a careful and deliberate observation is unfortunately often overlooked and underappreciated, although important, since it may provide clues about the condition of the patient and the underlying disease process. Physicians from the mid- to late eighteenth century used their physical examination skills of observation viewing the venous pulses, chest wall, and abdominal movements during the cardiac and respiratory cycle as a method to diagnose pericardial effusion or adhesive pericarditis. Although observation seems like a simple technique, it in fact requires training, patience, perceptibility, and practice. These observations, recognized as medical eponyms, honor those physicians who initially reported these signs. In the second part of the three-part series on inspection of the pericardium, the history of physicians who observed the sign as originally described and the sensitivity, specificity, and predictive value of its application in modern-day clinical practice are summarized. The signs are presented chronologically, based on the year that they were first reported.

METHODS

PubMed, Medline, online Internet word searches, and bibliographies from the source text and textbooks sources were used. PubMed was searched using the Medical Subject Heading of the name of the eponym and text words associated with the sign.

Friedreich Sign

Nikolaus Friedreich (1825–1882) was born in Würzburg, Germany, studied medicine in Würzburg in 1844 and Heidelberg in 1847, and received his doctorate in 1850 (1, 2). In 1853, he habilitated as *Privatdozent* (adjunct professor) for special pathology and therapeutics and in 1857 was appointed Professor Extraordinarius of Pathologic Anatomy in Würzburg, a position formerly held by Rudolf Virchow (2, 3). In 1858, Friedreich was appointed Director of the Medical Clinic, Professor and Chair of Pathology and Therapeutics, and Professor Ordinarius at the University of Heidelberg (2). As a student of Rudolf Virchow in Würzburg, Friedreich was profoundly influenced by his approach to medicine founded on the principles of cellular pathology and pathological process. His area of special interest was in clinical diagnostics. In collaboration with Kekule, Friedreich determined that amyloid (term coined by Virchow) was a proteniaceous substance (2). His name is perhaps best recognized and associated with his work on hereditary spinal ataxia (Friedreich's ataxia), as described in his paper titled "Ueber Ataxie

mit besonderer Berücksichtigung der hereditären Formen” (“About Ataxia With Special Consideration of the Hereditary Forms”) published in *Virchow's Archives* in 1877. As a description of Friedreich's scientific approach:

All the works of Friedreich, however, the smallest as well as the most extensive, have a common and characteristic character; built on the firm foundation of pathological anatomy, they are distinguished by a universal historical and literary knowledge, by an all-encompassing comprehension and control of the substance, by the thoroughness and clarity of the representation. (1, p. 973).

What follows is a description of his character, as formulated by Kussmaul:

Seeing the young clinician in his hospital was a useful thing and a pleasure at the same time. For him, the activity there was not duty and work, but pleasure and enjoyment; the clinic was not merely a scientific workshop, but also a matter of the heart. You could not be gentler, more humane with the sick, than Friedreich, his mild, quiet dignity and safe action brought comfort and confidence to all, and for some it was a cure. In the technique of physical examination, there was no greater master. No one came close to him in the art of percussion. One could find no better percussion hammers or plethimeters than Friedreich's fingers. Even on the most corpulent individual, he lured the notes far and wide. (2, pp. 203–204).

Friedreich reported initially in his chapter “Die Krankheiten des Herzens” (“The Diseases of the Heart”) in *Virchow's Handbuch der speciellen Pathologie und Therapie (Virchow's Handbook of Special Pathology and Therapy)* in 1861, a “peculiar phenomenon of the cervical veins which he interpreted as a new sign for the diagnosis of cardiac adhesions (4).” In his paper titled “Zur Diagnose der Herzbeutelverwachsungen” (“For the Diagnosis of Pericardial Adhesions”) published in 1864, Friedreich stated:

Since then not only the case to which I referred at that time attained at necropsy, but also a second case has been found in my observation, in which, in the presence of the same symptoms, the diagnosis of pericardial obliteration was confirmed during life in the case of the same subject, so that I can now consider the previous only with probability-pronounced sentences as sufficiently well founded, and believe [that I] can attach a new and essential sign to the still somewhat uncertain diagnosis of the pericardial adhesions. At the same time, the two cases to be described appear to me to be suitable for rectifying and modifying some other points concerning the diagnosis of pericardial adhesions. (5, pp. 296–297).

[T]he two cases presented here, which in many respects agree with each other, seem so instructive in the diagnosis of pericardial dilatation that their more

specific communication, as well as a closer examination of the phenomena observed during the course of the disease, may well be justified. (5, p. 304).

He explained and proposed a pathogenesis for the occurrence of systolic retraction and diastolic recoil of the chest and later its effects on the jugular vein reasoning in both cases:

There seems to me to be no doubt that such widespread systolic retraction must occur particularly when the heart shortens in the longitudinal axis when the systole pulls the diaphragm strongly upwards, and thus the points of insertion of the diaphragm on the chest, inward, whose motion also increases the adjacent higher portions of the thoracic wall, which are obliged to follow to a certain extent. After all, the occurrence of this phenomenon seems to be much favored by the more intimate adhesions of the lower surface of the heart with the diaphragm, which existed in an unusually strong manner, especially in the second case I have described. It seems probable to me that the latter phenomenon occurs only in cases with such fixed adhesions to the diaphragm, even without the obliteration of the remainder of the pericardial cavity. Thus, this prevents the downward sliding motion of the heart on the diaphragm, causing the diaphragm to be pulled upward by the systolic shortening of the heart. In contrast, if there are no diaphragmatic adhesions, despite existing pericardial adhesions in the upper parts of the heart, the above phenomenon may well be missed by freeing up the space caused by systolic shortening and elevation of the heart either by the expansion of the lung or even by a slight upward movement of the diaphragm. As a result of the pressure of the intestinal gases, the shortening of the apex of the heart could at most be marked by a systolic, circumscribed depression in the intercostal space in question. The remarkable fact that the systolic retractions of the chest wall were particularly pronounced at the level of the inspiratory movements may also be used to support the view presented, as the tighter, flattened, and shortened diaphragm had to be pushed higher, especially with deep inspirations, and the visual effect is more noticeable than during the flaccid and curved expiratory position of the diaphragm (...) Apart from the diaphragmatic adhesions, a second moment seems to require extensive systolic retraction of the chest wall, namely, an unbroken and sufficiently strong energy of the cardiac contractions, which has the task of overcoming the elasticity of the thorax, which certainly provides a considerable resistance. (5, pp. 307–309).

Friedreich described the appearance of the jugular veins during systole and diastole:

Simultaneously, with the diastolic recoil of the chest wall, a very clearly visible, sudden, swelling, alternating with the carotid pulse, followed by a rapid and

Table 1. Inspection and pericardial eponyms signs from 1864 to 1895

Name	Year	Description of sign	Significance
Friedreich	1864	During diastole, there is a rapid and brief jerking and venous collapse seen best at the anterior jugular vein. During systole, the veins become gradually lifted and filled.	Adhesive pericarditis
Duchenne	1883	During inspiration, the epigastric and hypochondriac regions are depressed, and the chest dilates. During expiration, these movements are reversed. When the diaphragm is weak, these signs are seen only during forced respiration.	Pericardial effusion
Broadbent	1895	Retraction synchronous with the cardiac systole occurring posteriorly in the region of the left and right eleventh and twelfth ribs. This is most marked on the left compared to the right side.	Adherent pericardium

brief jerking diastolic venous collapse could be seen, and in one of the cases involved the entire supra-clavicular region which in both the right and left side was recessed and pulled inwards with a quick, short movement. In both cases, this diastolic collapse was best seen in one of the right infraclavicular regions from the bottom to the top, and extending downwards just below the clavicle and externally at the greater skin-vein, especially recognized at the anterior jugular vein. During the systolic retraction of the thoracic wall, the veins were again gradually lifted and filled to a not insignificant degree (in the second case with a double motion, corresponding to the contraction of the atrium and ventricle), and at the moment of diastole to collapse again to complete disappearances. These described phenomena of the veins were so peculiar and striking that they could not be confused with any of the more common occurrences of the jugular veins. Evidently the emptying of the veins at the moment of ventricular diastole must have been favored to a very special degree, and it could be understood very well from the prevailing circumstances. (5, pp. 310–311; emphasis added).

Furthermore, he accounted for the occurrence of this finding in adhesive pericarditis:

To a certain extent the sudden decreased pressure in the lungs produced by the inward movement of the chest wall may be conducive to a rapid outflow of blood from the jugular veins. This may well be compared to the rapid diastolic recoil of the chest wall and retarding influences of the previous systolic retraction to have a similar effect, with such a rapid and deeper inspiratory movement, in the jugular veins. Moreover, the reason for the above appearance seemed to be attributed to a change of position of the diaphragm caused by the recoil of the chest wall. As the result of the return of the ribs and the lower part of the sternum into a deeper and flatter position, it was necessary for the heart, which had adhered to the diaphragm, to undergo a movement during diastole, contrary to the conditions prevailing under normal conditions. (5, p. 311).

He acknowledged work by other authors in this field of study:

The phenomenon described here of a rapid diastolic collapse of the cervical veins swelling gradually in systole has already been mentioned by other observers, and Skoda regards this rare symptom as a sign of paralysis of the right atrium, though it is difficult to see how it should arise in the latter state. As a sign for pericardial obliteration, however, I find the phenomenon not described or listed by any of the authors known to me. (5, p. 312).

Thus, the Friedreich sign refers to diastolic collapse of the cervical veins in patients with adherent pericardium (Table 1). In a study of 15 cases of constrictive pericarditis and 17 normal controls, diastolic venous collapse was identified in 8 of 15 cases (6). Lang et al. identified the Friedreich sign in 15 of 16 patients with constrictive pericarditis. It is generally absent in cases of cardiac tamponade as it is more often associated with a restrictive and heavily calcified pericardial disease (7).

Duchenne Sign

Guillaume-Benjamin-Amand Duchenne (1806–1875) was born at Boulogne-sur-Mer (Boulogne), France, studied medicine in Paris where he was taught by eminent physicians such as Cruveilhier, Velpeau, Dupuytren, and Laënnec, and graduated in 1831 (8, 9). Subsequently, he practiced in Boulogne, returning to Paris as a private practitioner in 1842 where he worked in Pierre-François Olive Rayer, Armand Trousseau, and Jean-Martin Charcot at Charité, Hôtel-Dieu, and Salpêtrière hospitals, respectively (8, 9). Both Trousseau and Charcot were influential in that they encouraged Duchenne to publish his discoveries (8). Despite not having an appointment or position at the University, he remained committed to clinical neurology research. He described his position related to this matter in his book titled *De l'électrisation localisée et de son application à la pathologie et à la thérapeutique (From Localized Electrization and Its Application to Pathology and Therapeutics)* in response to criticism he received related to his failure to address the study of pathological anatomy during his clinical studies of glosso-labial-laryngeal paralysis:

I must answer these reproaches that I do not think I have deserved. Those who made it to me probably did not know that I was only allowed to glean, so to speak, from our hospitals, and that if I have been able to exist scientifically, it was because I have been fortunate enough to collect, having browsed through the many services of hospital, the clinical

facts, which have been, I will not say disdainful, but which had escaped the observation of all. Those who criticized me should have known that when I discovered a morbid species not yet described, I was not allowed to discuss subjects that I had made during my lifetime clinical research. I would have had, it is true, the freedom to complete these by anatomopathological research, if I had been a hospital doctor. But then, I could not have fulfilled the task of researcher that I had imposed on myself, and I am convinced that my principal work (among other things, progressive musculo-atrophy, atrophic paralysis of childhood, progressive locomotor ataxia, glosso-labio-laryngeal paralysis and pseudo-hypertrophic paralysis) would never have seen the light of day. To accomplish them, I needed a wider field of observation, spanning all the hospitals in Paris. If, therefore, I have renounced, and still renounce, the honors of an official positions, it is because of my duty to give myself entirely and freely to my irresistible taste for physiological and pathological research. Should I be a subject of reproach for the disinterestedness of having made science solely out of love for it? (10, p. 601).

Duchenne noted that there are different causes of diaphragmatic paralysis including the one caused by “palsies due to the inflammation of neighbouring organs” (11, p. 329). He further described the symptoms that occur in these conditions are

[n]oticeable during respiration, and are as follows: At the moment of inspiration the epigastric and hypochondriac regions are depressed although the chest dilates; during expiration these movements are reversed. When the diaphragm is merely weak these signs are seen only during forced respiration, but during tranquil breathing the bulging of the belly and the expansion of the chest, and vice versa, occur together, as in health. (11, p. 330; emphasis added).

Thus, the Duchenne sign refers to the presence of depression of the epigastrium and hypochondriac regions in patients with diaphragmatic paralysis and occasionally in pericardial effusion, although we were unable to confirm that he described the latter specifically in relation to pericardial effusion (12) (Table 1). Duchenne made numerous discoveries and contributions to the medical literature with eponymous conditions and diseases that bear his namesake, including the Duchenne attitude, Duchenne disease (Tabes dorsalis, progressive bulbar palsy), Duchenne paralysis, Duchenne–Aran disease, Duchenne Erb–Palsy paralysis, and Duchenne–Griesinger disease (Duchenne muscular atrophy or dystrophy) (8,13). We are unaware of any studies that evaluated the sensitivity or specificity of this sign.

Broadbent Sign

William Henry Broadbent (1835–1907) was born at Longwood, near Huddersfield, England, passed the qualifying examination in 1857 from the College of Physicians, London, and received his medical degree from the University of London in 1860 (14, 15). He served at St. Mary’s Hospital in 1858, London Fever

Hospital in 1860, and Western General Dispensary, as an outpatient, full, and later as a consulting physician (16). He was a member of the Royal College of Physicians in 1861, fellow in 1869, censor 1888–1890, and senior censor 1895–1896 (15, 17). He was appointed President of the Harveian Society in 1863, Medical Society of London in 1881, Clinical Society from 1887 to 1888, and Neurological Society from 1895 to 1896 (16, 17). In 1897, he was elected fellow of the Royal Society and received the honorary titles of LLD from the Universities of Edinburgh in 1896, St. Andrew in 1897, Toronto in 1906 and D.Sc. from the University of Leed in 1904 (15, 17–19). He is the recipient of a number of distinguished honors, including the Knight Commander of the Victorian Order in 1901, Grand Cross and Insignia of the Legion of Honour in 1904, and physician extraordinary to Queen Victoria and physician-in-ordinary to King Edward (15, 17, 18). His work spans primarily the fields of cardiology and neurology and additionally in the areas of cancer and typhoid (19).

In tribute to Broadbent’s character, a former student wrote in an obituary the following description:

Those who kept in touch with him could always be sure of kindly and wise counsel for the asking, whether the matter were one as to a career, a perplexing case, or personal or domestic illness. In the first two of these contingencies I have myself repeatedly experienced his unfailing kindness; in the last I have known of instances where all the engagements of a busy life have been put aside to make time to give freely his aid in consultation at the bedside of some former pupil, not only in or near London, but at distances involving a railway journey of many hours each day. (17, p. 71).

William Broadbent described in 1895 in his paper titled “An Unpublished Physical Sign” the finding identified in four patients of a

[v]isible retraction, synchronous with the cardiac systole, of the left back in the region of the eleventh and twelfth ribs, and in three of which there is also systolic retraction of less degree in the same region of the right back. In all the cases there is a definite history of pericarditis, and in three of them there are other conditions strongly suggesting an adherent pericardium. (20, p. 200; emphasis added).

Furthermore, he discussed the findings of adherent pericardium at the Medical Society’s Transaction in 1898:

A systolic tug of the left false ribs posteriorly communicated by the diaphragm may be conspicuous. The recoil from the drag may be so distinct as to look and feel to the hand like pulsation, and in the first case in which I observed it, now more than 20 years since—a case of left empyema—it was taken for pulsation, and it was supposed that a pulsating tumour of some kind underlay the empyema. A post-mortem examination showed that the cause was adherent pericardium. (21, p. 25; emphasis added).

He accounted for the proposed pathogenesis of this occurrence due to the

[d]iaphragm, which, if pulled upon, would have more effect on the floating eleventh and twelfth ribs than on the other more fixed ones. In cases of large heart with adherent pericardium there is a considerable area of the ventricles closely adherent to the central tendon of the diaphragm, and the powerful contraction of the hypertrophied heart must give a decided tug to this structure. That it should affect the ribs more often on the left side would be expected from the adhesion being mainly to the left of the middle line; the liver also, which is often large in these cases, may restrain the movement to the right. (22, p. 200).

He believed that “*systolic retraction of the lower ribs on the lateral or posterior aspect of the thorax is of the greatest importance, and can scarcely be due to anything else than pericardial disease*” (22, p. 56; emphasis added). Broadbent sign thus refers to the systolic retraction of the chest wall near the eleventh and twelfth rib posteriorly due to pericardial adhesions (Table 1). We are unaware of any study that evaluated the sensitivity or specificity of this sign.

CONCLUSION

From 1864 to 1895, physicians continued to observe signs related to protrusion and retraction of the chest wall during cardiac contraction and respiration. Friedreich’s observation is notable as it provided insights of events occurring in the jugular veins that correlate with the cardiac cycle. An appreciation of these signs is important as it assists clinicians to understand the pathophysiologic mechanism of pericardial disease.

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