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Physical Signs of Inspection and Medical Eponyms in Pericarditis Part III: 1900 to 1964

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ABSTRACT

During the 20th century with emerging technological advancements including the development of fluoroscopy, radiography, and electrocardiography, physicians used these tools in patients with acute or chronic pericarditis with or without pericardial effusion to identify signs eponymously named to honor the person who first discovered, described, and reported the discovery. Hence, through meticulous observations, these signs complimented other findings found on physical examination to further assist in supporting the diagnosis of pericardial disease.

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

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INTRODUCTION

During 1900–1964, physicians continued to use their keen sense of sight to identify abnormalities on physical examination, radiographs, and electrocardiogram to diagnose pericarditis. Inspection is the first basic tenet routinely taught during physical examination. It is often discarded as being insignificant and thus only given cursory significance. It is the method used for identifying physical sign by observation (1). As emphasized by DeGowin and DeGowin in their book *Bedside Diagnostic Examination*, “More diagnoses are probably made by inspection than by all other methods combined. The method is the most difficult to lean because no systematic approach can encompass the variety of signs” (1, p. 34). “Inspection,” although technically used in reference to physical examination, is the medical term applied in the present study when “observing” a sign detected through radiography or electrocardiogram. Physicians identified other diagnostic signs that further aid in supporting the diagnosis of pericarditis using radiography and electrocardiography. We briefly described the person who initially reported the sign as originally described and if available, the sensitivity, specificity, and predictive values in this final series on the signs of inspection of the pericardium in patients with acute, adhesive, and constrictive pericarditis. These signs are presented chronologically based on the date they were first published during this period.

METHODS

PubMed, MEDLINE, online Internet word searches, and bibliographies from text and textbook sources were used. PubMed was searched using the Medical Subject Heading of the name of the eponyms and text words associated with the sign.

Ogle Sign

Cyril Ogle (1861–1931) was born near Sheffield, UK. He attended medical school at St. George’s Hospital beginning in 1883, qualified in 1888, and was appointed assistant in 1897, physician in 1904, and consulting physician in 1926. He was elected member of the Royal College of Physicians, London in 1891 and fellow in 1899 (2). He was active in the chapter serving as an examiner in pharmacy, medicine, councilor, and censor and was Vice President of the Clinical Section from 1922 to 1927 (3). H.R. wrote that “His exceptional abilities were fully recognized by his colleagues and pupils, who paid him the well-deserved tribute of making him the doctors’ doctor. A more willing and unselfish friend cannot be imagined, and it is certain that he spent his life in the service of others” as a tribute to his character (3, p. 426).

He and Herbert Allingham described a patient with purulent pericarditis as “[b]oth external jugular veins were very distended, not altering during inspiration, and without visible pulsation or even flicker, but they contained liquid blood.” (4, p. 693).

Thus, the Ogle sign refers to the presence of distention of the external jugular veins without pulsation in patients with purulent pericarditis (Table 1). Non-pulsatile distended jugular veins or neck veins are found in cases of

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obstruction (e.g., constrictive pericarditis, mediastinitis, and thrombosis of the upper thoracic veins) or unilateral or bilateral compression (e.g., enlarged mediastinal or cervical lymph nodes and aortic aneurysm), resulting in impaired venous return to the right heart (5). The height of distension of non-pulsatile neck jugular veins, in contrast to pulsatile jugular veins, is neither affected by changes in upper body position from the level of the horizontal plane or respiration (5). Thus, this is a useful method for distinguishing the potential etiologies of this finding. The finding of distended neck veins in a wide variety of diseases suggests that it is a non-specific sign for the diagnosis of constrictive pericarditis.

Keith Sign

Arthur Keith (1866–1955) was born in Old Machar, Aberdeen, Scotland. He began his medical training at Marischal College in 1884 and graduated with an MB in 1888 (6, 7). He accepted a position as a medical officer in Siam, Thailand in 1889 where he also developed his interest in botany collecting over 500 flora specimens (8). He returned to London in 1891 to pursue his primary interest in comparative anatomy and paleontology. He received his medical degree at Aberdeen University and fellowship from the Royal College of Surgeons in 1894. He lectured on Anatomy at the London Hospital Medical School in 1895 where he was involved in the study of the anatomical bases of the mechanisms of respiration and cardiac function including cardiac arrhythmias with James Mackenzie and Martin Flack (7, 8). He and Flack collaborated to identify the sinoauricular (sinoatrial) node (7, 8). He was appointed Hunterian Professor and Conservator of the Hunterian Museum at the Royal College of Surgeons in England in 1908, was elected President to the Royal Anthropological Institute between 1914 and 1918, and was acting editor of the *Journal of Anatomy* from 1916 to 1933 (7–10). His other accolades included being appointed Fullerian Professor of Physiology of the Royal Institute of Great Britain from 1918 to 1923, elected Rector of Aberdeen University in 1930, and knighted in 1923 (8, 9). His life's work and career can be summarized into three facets:

He was, of course, primarily a human anatomist and his knowledge of human anatomy (particularly in relation to its clinical applications) was quite profound. Secondly he was a comparative anatomist having an unrivaled personal acquaintance with the morphology of the higher Primates. Thirdly, he was a physical anthropologist who allowed his interests to stray far beyond the narrow field represented by measuring tape and calipers (8, p. 155).

He was the recipient of six honorary degrees, such as the Legum Doctor from the University of Aberdeen, Doctor of Law from Birmingham and Leeds, and Doctor of Science from Durham, Manchester, and Oxford (7).

He described the phenomenon of lateral thoracic respiratory expansion in cases of pericardial adhesions:

When the pericardium becomes fixed by adhesions to the lungs and chest wall a number of obscure symptoms follow owing to the arrest of the normal respiratory movement of the heart. The type of respiration may change; the patient, instead of gaining his

inspiratory space by a forward heave of his abdominal wall, due to the descent of the diaphragm, inspires by expanding the walls of his chest and body in a lateral direction (11, p. 558; emphasis added). (...) In that movement the central part of the diaphragm and its crura take no part (11, p. 559).

To our knowledge, no other studies evaluated the sensitivity or specificity of this sign (Table 1).

Wenckebach Sign

Karel Frederik (Karl Friedrich) Wenckebach (1864–1940) was born in The Hague, Netherlands. He received his medical degree in 1888 from the University of Utrecht (12). He had an initial appointment as an assistant at the Institute of Zoology and subsequently in the Departments of Pathology and Normal Anatomy in 1888 (12). He served as Professor of Pathology and Therapeutics and Clinic and Director of Pathology Laboratories from 1901 to 1911 at the University of Groningen, the Netherlands (12, 13). He was appointed Chair of Medicine at the University of Strasbourg and University of Vienna from 1911 to 1914 and from 1914 to 1929, respectively. He was an honorary fellow and a foreign corresponding member of the British Medical Association in the Royal College of Physicians and Royal Society of Medicine (12). Sir Thomas Lewis amply described the extent of Wenckebach's studies in the field of cardiovascular disease:

When as a young man I first met Wenckebach it was at Groningen, where he then held the chair of medicine. He had already reached fame by his independent analysis of cardiac irregularities, using the radial pulse only; this work, published as whole in his first book, *Die Arrhythmie*, in 1903, was a brilliant achievement and the foundation of much that followed. (...) In later years, by publishing an instance in which quinine stopped attacks of auricular fibrillation, he made a first observation that led quickly to the successful use of quinidine for chronic fibrillation. His work upon beriberi heart, undertaken in post-war years in the Dutch East Indies, again broke fresh ground and must be regarded as of high importance (12, p. 219).

He showed by taking instant photographs during respiration in a patient with adhesive pericarditis, instead of the lateral expansion of the chest as described by Keith, during inspiration that there is:

[o]nly an upward movement of the chest and that very limited. There is no forward movement of chest wall whatever. Looking at the patient, we see an inspiratory retraction of the lower part of the sternum and ribs. (...) So we find in this disease a paradoxical respiratory movement of the chest, a pendant to the so called paradoxical pulse (14, p. 68; emphasis added).

He accounted for this finding that adhesions cause the diaphragm to:

[n]ot only pull at the heart, but pulls at the chest wall in front. (...) The absence of a marked descent of the diaphragm in inspiration is proved by the ascent

Table 1. Inspection and pericardial eponym signs (1900–1964)

Name	Year	Description of the sign	Significance
Ogle	1900	Distension of the external jugular veins unaltered during inspiration and without visible pulsation.	Purulent pericarditis
Keith	1904	Respiratory movements in patients with adherent pericardium. The patient, instead of gaining his inspiratory space by a forward heave of his abdominal wall due to the descent of the diaphragm, inspires by expanding the walls of his chest and body in a lateral direction.	Adherent pericardium
Wenckebach	1907	Respiratory movements of the chest wall in patients with adherent pericardium. There is only an upward movement of the chest that is very limited, as well as an inspiratory retraction of the lower part of the sternum and ribs. There is a paradoxical respiratory movement of the chest, a pendant to the so called paradoxical pulse.	Adherent pericardium
Hirtz	1911	Patient assumes a knee–chest position to relieve dyspnea. The patient is in bed on his/her knees, body bent forward, and supported by the elbows.	Pericardial effusion
Wynter	1911	Inhibition of the action of the diaphragm, indicated by the suppression of normal abdominal respiratory movement. The cessation of phrenic action is bilateral, and consequently the absence of abdominal movement is as obvious as in abdominal disorders, although other abdominal manifestations, such as muscular rigidity and general tenderness, are lacking in pericarditis.	Acute fibrinous pericarditis
Volhard	1923	Retraction of the heart and chest wall during systole and its return in diastole. Also known as Brauer’s diastolic chestnut sling.	Mediastinal pericardial adhesions
Schmieden	1926	Radiographic and operative finding of systolic retraction of the left leaf of the diaphragm in cases of constrictive pericarditis.	Constrictive pericarditis
Rabin	1952	With the neck in a neutral position, the elevation of the aortic arch on swallowing indicates an abnormal adherence of the aortic arch to the trachea. This is called the <i>aortic swallowing sign</i> .	Mediastinal pericardial adhesion
Spodick	1964	The predominant P–R segment depression across the pericardium documents the posterior component. Owing to these P–R segment deviations, it is of practical importance in acute pericarditis to consider the T–P segment to be the electrocardiographic baseline to avoid mistaking P–R segment depression for S–T segment elevation.	Acute pericarditis

of the vaulting of the abdominal wall. Even we see an inward movement probably due to the fact that the chest with the adherent diaphragm is strongly heaved off from the abdominal contents (14, p. 66).

He thought that:

[i]t is a very valuable sign for the diagnosis of adherent pericardium (14, p. 65). (...) So every new symptoms has to be welcomed, and I venture to think that the arrest or even retraction of the lower part of the sternum with its cartilage in inspiration *must* be found in every case whether there are adhesions between diaphragm, heart, and chest wall (14, p. 67).

Francis Sibson described in 1881 the finding of limited anterior chest wall expansion in his chapter on adherent pericardium:

When, however, the heart is, as usual, enlarged, being often affected with valvular disease, the adhesions may be short, fibrous, and binding; and the front of the organ may be fixed to the two lower thirds of the sternum and the adjoining cartilages by pleuropericardial adhesions, so that the automatic

and respiratory movements of the heart, and the inspiratory expansion of the lungs, are restrained; thus the discovery of the adhesions during life may generally in such cases be made by a careful study of the physical signs; its diagnosis being the more certain and easy in proportion as the heart is more enlarged, and more firmly fixed to the anterior walls of the chest (15, p. 203).

Thus, the Wenckebach sign refers to the absence of the anterior movement of the chest during inspiration in patients with adhesive pericarditis (Table 1). To our knowledge, no other studies evaluated the sensitivity or specificity of this sign.

Hirtz Sign

Edgard Hirtz (1849–1916) was born in Winzenheim, Haut-Rhin, Alsace, Germany. He received his medical training at the old faculty of Strasbourg and Paris. He was appointed physician at the Hôpitaux de Paris in 1887, head of the Department of Necker Hospital Paris, and officer of the Legion of Honor in 1910 (16–18).

He described in his paper entitled “Knee chest position as a sign of pericarditis” the findings in a patient with acute generalized articular rheumatism and dyspnea:

He was obliged to sit up, leaning forward for a few minutes at a time, but even this position did not give him the desired relief. *The patient was relieved of his dyspnea only when he assumed the knee–chest position, which he instinctively took himself. It is this singular position which attracted my attention when I first saw the patient. He was upon his knees on the bed, his body bent forward, and supported by his elbows.* The first sight of this individual in a condition of intense anxiety, cyanosis, and very short of breath, bent forward in the knee–chest position made me think of the possibility of pericarditis with marked effusion, even without proceeding with the examination (19, p. 122; emphasis added).

Based on his observations and the improvement of dyspnea with thoracentesis:

I believe, in fact, that the knee–chest position necessitated by an intense dyspnea is characteristic sign of pericarditis with large effusions. While this sign is rare, it seems to have a definite value; for in my experience it always indicates the presence of a large pericardial effusion (19, p. 123).

Thus, the Hirtz sign refers to the improvement in the symptom of dyspnea when the patient assumes the “knee–chest posture” (Table 1). This sign is also found to be helpful in patients with “rheumatic, tuberculous, post scarlatinal and nephritic pericardial effusions” (20, p. 49). He acknowledged that Zehetmayer in 1845 and Merklen in 1892 each reported a case where a patient with large pericardial effusion assumed the knee–chest position (19).

Wynter Sign

Walter Essex Wynter (1860–1945) was born in Brompton, London, England. He received his medical training at Middlesex and St. Bartholomew Hospital, London and medical degree in 1888 (21). He served as assistant in 1891, full physician in 1901, and consulting physician in 1925 at Middlesex Hospital (21). He was elected Fellow of the Royal College of Physicians in 1897 (22). In addition to the sign that bears his namesake, it is of historical interest that he reported in 1891, the same year, but 1 month after Heinrich Irenaeus Quincke performed the first lumbar puncture. His technique differed from Quincke in that it involved an incision to the dura followed by insertion of a Southey’s tube with rubber drainage at the L2 intervertebral space rather than a very fine cannula between the third and fourth lumbar vertebral arches (23, 24). He described a sign that he identified in patients with acute fibrinous pericarditis secondary to rheumatic heart disease with predominately polyarthritis:

The sign suggested in this communication is inhibition of the action of the diaphragm, indicated by suppression of normal abdominal respiratory movement. (...) [t]he cessation of phrenic action is bilateral, and consequently absence of abdominal movement is as obvious as in abdominal disorders, albeit other abdominal manifestations, such as muscular rigidity and general tenderness are lacking (25, p. 40; emphasis added).

He recognized that “inflammation of the pericardium, possibly commencing in some other part than that immediately in contact with the chest wall, indicated by its presence by reflex inhibition of the diaphragm some days before the appearance of ordinary sign” (25, p. 42). Thus, “Loss of abdominal movement, as a sign of pericarditis is the more valuable, in that it may precede and outlast the other indications, and so afford grounds for anticipatory or retrospective diagnosis” (25, p. 45). He acknowledged and referred to the work in Sibson’s textbook *Collected Works of Francis Sibson* published in 1901 where this finding was described:

(...) The respiratory play of the upper ribs was more than doubled in extent in three-fourths of the cases observed (5 of 7), so that respiratory was as a rule high. This was due to the arrest or restraint of the action of the diaphragm caused by extensive inflammation of the central tendon of the diaphragm, where it forms the floor of the pericardium (26, p. 141; emphasis added).

Furthermore, Sibson accounted for the proposed pathogenesis of this finding:

The inspiratory movement of the abdomen below the ensiform cartilage was either reversed (in 12), arrested (in 1), or restrained (in 6) in every case of pericarditis in which that sign was observed. This is at once accounted for by the inflammation, in that disease, of the central tendon of the diaphragm where it forms the floor of the pericardium, which leads to the virtual paralysis of the central portion of the diaphragm. This fact, that the anterior wall of the epigastric space, instead of advancing, recedes during inspiration, gives us a physical sign of great value in the diagnosis of pericarditis, and of the advance and decline of that disease (26, p. 142).

Thus, the Wynter sign refers to the absence of respiratory abdominal movements in patients with adhesive pericarditis (Table 1). To our knowledge, no other studies evaluated the sensitivity or specificity of this sign.

Volhard Sign

Franz Volhard (1872–1950) was born in Munich, Germany. He received his medical education at the Universities of Bonn, Strasbourg, and Halle and completed his State Examination in 1897 from the University of Halle (27, 28). He worked as a morbid anatomist followed by a position as a lecturer at the University of Gießen in 1898 in Berlin (27, 29, 30). He served as Director of the Clinic of Internal Medicine at the Dortmund Municipal Hospital in 1905 (29, 31). He was the Director of the Municipal Hospital in Mannheim in 1908 and Chief of Internal Medicine in 1910–1918 and appointed Professor and Director of the Clinic of Internal Medicine at the University of Halle in 1918 (29, 30). He accepted the position as Professor of Medicine at Frankfurt am Main in 1928 and was President of the German Society of Internal Medicine in 1930 (28, 30).

He received an honorary doctorate from Sorbonne (University of Paris) in 1933 and was the co-editor of *Zentralblatt für innere Medizin* and chief editor of *Neue medizinische Welt* (31, 32). His

research expanded upon multiple therapeutic areas including nephrology (hypertension, low-protein diet in uremia, clinical aspects of uremia, and isosthenuria marker of chronic renal disease), pulmonary (positive pressure respirator and method to assess peak flows at the bedside in bronchospastic and emphysematous lung disease), cardiovascular (increased pansystolic venous pressure in pericarditis and retinal findings or retinitis vasospastic of the vessels in patients with severe hypertension with proteinuria), infectious diseases, neurology, and gastroenterology (identified and characterized gastric lipase) (29, 30, 31). He is perhaps best recognized for his work on diseases of the kidney. He and Theodor Fahr, his colleague, in their monograph *Die Brightsche Nierenkrankheit: Klinik, Pathologie und Atlas* (Bright's Kidney Disease: Clinic, Pathology and Atlas) published in 1914 revolutionized the field of nephrology by classifying renal disease (Bright's disease) into three main groups: inflammatory diseases that involved the glomeruli or glomerulonephritis (nephritis), degenerative disease or nephrosis, and arteriosclerotic (sclerotic) renal disease (29, 30, 32). Their work was influential in distinguishing benign from malignant nephrosclerosis forms of hypertension. With regard to the extent of his contribution to nephrology:

It remains true, however, that no one since Bright has done more than Franz Volhard to advance knowledge in this important branch of medicine. For more than a generation his clinic at Frankfurt was a Mecca for those interested in renal disease, and his teaching and methods for long prevailed throughout the civilized world (32, p. 1376).

With Viktor Schmieden, his colleague, they reported their observation of the motion of the chest wall in a patient with calcific pericarditis with adhesions:

It is possible to distinguish two type of callous pericarditis depending on whether external adhesion of the decay pericardium to the chest wall or the shrinkage of the callous thickened pericardium dominates the clinical picture. The difference is that in the one case in which the mediastinal factor outweighs the soldering of the heart to the chest wall, the heart becomes overtaxed during systole, for at each contraction it must overcome the resistance of the relatively rigid chest wall by increasing the pull inward to the left. *In the other case, in which the callous clasp of the heart predominates, diastole is primarily effected. In the first case we see the picture of a more or less profound weakness of the heart muscle, the universality of the pulmonary margins and the heart at respiration and at the positional plate, and above all, the characteristic retraction of the heart and chest wall at every systole and its return in diastole. Brauer's diastolic chestnut sling, which is only too easily mistaken for a top bump* (33, p. 5; emphasis added).

His name is only ascribed to this physical finding. By convention, since both names appeared on the publication, it would be more commonplace to refer to this sign as Volhard-Schmieden sign in recognition of the contributions by both authors. Thus, the Volhard

sign has been referred to as the “see-saw” movement of the chest (Table 1). It is the systolic displacement of the thorax to the right with an opposite (leftward) movement in diastole (34). They recognized that this sign is not pathognomonic of adhesive pericarditis and identified it in cases of severe mitral stenosis:

Yes, even here we often see a systolic constriction of the heart in the region of the right ventricle, which lies adjacent to the chest wall, and a swinging movement of the chest wall, which reminiscent of a chest wall spin of mediastinopericarditis. But instead of the silence over the heart, we feel the moderately lifting action of the right ventricle in the region of the cardiac dullness and below the left costal arch, and hear the characteristic mitral rhythm and the diastolic sound above the tapping tip (33, p. 6).

This suggests that the sign presumably lacks sufficient specificity when used alone in the diagnosis of adhesive pericarditis.

Schmieden Sign

Victor (Viktor) Gottfried Otto Schmieden (1874–1945) was born in Berlin, Germany. He received his medical training at the Universities of Freiburg, Munich, Berlin, and Bonn and medical doctorate from the University of Bonn. He was a trainee assistant at the Pathological Institute of the University of Göttingen from 1898 to 1900. He habilitated for surgery in Bonn in 1903 and was a *Privatdozent* (adjunct professor) in 1907 (35). He was appointed Professor of Surgery at Friedrich-Wilhelms-Universität, Berlin between 1908 and 1913 and Professor and Chair of Surgery at the University of Halle from 1913 to 1919 and served as second Professor of ordinary for Surgery at Frankfurt University am Main in 1919 (35, 36). He was also a member of the German Academy of Sciences Leopoldina in 1916 (35).

He made a number of contributions to medicine including laparotomy for the management of abdominal gunshot wounds and pericardiectomy for calcific pericarditis (Schmieden's operation) and recognized the sequence from adenomatous polyps to colorectal cancer (dysplasia–carcinoma) recommending total colectomy for patients with polyposis coli (37, 38).

In addition to the sign that he reported with Volhard, he also described the fluoroscopic appearance of the diaphragm in cases of constrictive pericarditis caused by adhesion between the pericardium, mediastinum, and pleura. In this case, there is systolic elevation of the diaphragm due to attachments between the diaphragm and the cardiac apex (39). Interestingly, he also described this phenomenon intraoperatively:

After the lateral and posterior surface of the left ventricle is free, the diaphragmatic surface and the apex are separated with sharp and blunt dissection. The result of this is very impressive. *The diaphragmatic portion of the callus surrounds this section of the heart like petals. The apex, bound down to the diaphragm, cannot produce the normal systolic retraction. On the contrary it becomes elongated during the systole and ends in a sharp one.* After this section has been freed, the apex regains its normal systolic retraction (38, p. 91; emphasis added).

Thus, the Schmieden sign is a radiographic finding in patients with pericardial adhesions where there is visualized, systolic retraction of the left leaf of the diaphragm occurring during inspiration (34) (Table 1).

Rabin Sign

Coleman Berley Rabin (1900–1991) was born in New York City, USA. He received his medical degree from Columbia University College of Physicians and Surgeons in 1922 (40). He was an intern at Mount Sinai Hospital in 1922 where he completed training in the pathology laboratory from 1927 to 1938 (41). He was Clinical Assistant Professor of Medicine at Mount Sinai School of Medicine, elected President of the American College of Chest Physician, and recognized for his professional achievements in medicine by receiving the Townsend Harris Medal by the Associated Alumni of the College of the City of New York (41, 42).

As described by Benjamin Felson in chapter 24 entitled “Some special signs in roentgenology” in Rabin’s book *Roentgenology of the Chest* published in 1958:

With the neck in a neutral position, elevation of the aortic arch on swallowing indicates an abnormal adherence of the arch to the trachea. Rabin has called this the *aortic swallowing sign* (43, p. 259–260).

Faber noted that this finding may be found in patients with pulmonary emphysema, luetic aortitis, aneurysm, mediastinal tumor, and adhesive pericarditis and typically identified as a sign in constrictive pericarditis. As to the pathogenesis of this sign, it is believed to be caused by increased tension of the paratracheal connective tissue in the case of severe pulmonary emphysema when the heart and aorta assume a downward displacement and when the elongation of the aortic arch results in the connective tissue between the aorta and trachea to tighten (43). Thus, the Rabin sign or “aortic swallowing sign” refers to the presence of elevation of the cardiopericardial silhouette detected fluoroscopically on swallowing due to external pericardial adhesion originally reported in 1952 in Rabin’s book *Radiology of the Chest: Diagnostic Roentgenology* (44) (Table 1). The presence of this sign in other diseases suggests that it lacks sufficient specificity for the diagnosis of adhesive pericarditis.

Spodick Sign

David H. Spodick (1927–present) was born in Hartford, Connecticut, USA. He graduated from New York Medical College in 1950 (45). He completed an internship at St. Francis Hospital, Hartford, Connecticut and residency at Beth Israel Hospital and New England Medical Center (45). He served at Lemuel Shattuck Hospital for 19 years followed by a position of Chief of Cardiology at St. Vincent Hospital, Worcester, Massachusetts after completing advanced fellowship training in cardiology. He is Director Emeritus of the Cardiovascular Medicine Fellowship Program at St. Vincent Hospital and Professor of Medicine Emeritus at the University of Massachusetts Medical School (46). He published extensively and is the recipient of many accolades including the Melvin L. Marcus Memorial Award by the International Academy of Cardiology at the 3rd World Congress of Heart Disease for his distinguished contribution as a gifted teacher in cardiology (46). Gordon Saperia wrote “David Spodick has reminded us all that we should not be

too certain of what we know and the open mindedness may be our greatest intellectual asset” as a testimony to his character (45).

He retrospectively studied the electrocardiograms of 50 clinically diagnosed cases of acute pericarditis. He noted in his study “The T–P interval was the base line for P–R and S–T changes” (47, p. 471) and, “*The predominant PR segment depression across the pericardium document the posterior component. Because of these P–R segment deviations, it is of practical importance in acute pericarditis to consider the T–P segment to be the electrocardiographic base line to avoid mistaking P–R segment depression for S–T segment elevation*” (47, p. 474; emphasis added).

Thus, the Spodick sign refers to the presence of S–T segment elevation and P–R depression above or below the isoelectric T–P segment (Table 1). Based on this point of reference, S–T segment elevation was found in 48 and P–R depression in 41 (82%) of 50 patients (47). P–R segment deviation was found in 14 of 19 patients with acute pericarditis and absence of the typical findings of ST segment deviation (48). Kranin emphasized the importance of physical examination in patients with acute pericarditis. In their study, diagnostic electrocardiographic changes were identified in only 1 of 31 patients with a pericardial friction rub (49).

CONCLUSION

As we have found in prior series, in most cases, the signs have not been studied; thus, their applicability in clinical practice is unknown. The one major exception is the Spodick sign. The Spodick sign is the electrocardiographic finding of P–R depression using the isoelectric T–P segment as the baseline to diagnose acute pericarditis. Physicians continue to search for this finding as a marker to further confirm their suspicion for this disease. We believe that this series on the inspection of pericarditis provides insights and re-emphasizes the importance of this initial aspect of physical examination in the diagnosis of disease. We recognize that patients are more likely to present early in the course of the disease. Thus, these signs found in patients with chronic or adhesive pericarditis are less likely to be present. Nevertheless, knowledge of these signs is important since they provide an explanation of the pathogenesis and pathophysiology of the disease and a greater appreciation for the importance of this aspect of physical examination in diagnosis. They represent the window to better learn and provide a more profound appreciation for clinical medicine.

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