Where are the Diseases of Yesteryear?

DaCosta's Syndrome, Soldiers Heart, the Effort Syndrome, Neurocirculatory Asthenia — And the Mitral Valve Prolapse Syndrome

DACOSTA DESCRIBED "IRRITABLE HEART" in 1871,1 a peculiar form of functional disorder of the heart seen in the military population during the War Between the States. He thought this disorder was similar to that described earlier among British troops in India and in the Crimean War. The disorder frequently presented either after an episode of diarrhea and persisted after the digestive disturbances had passed away, or originated suddenly without previous digestive disorder. Two-thirds of his 300 patients were 16 to 25 years old. These cases represented the most common cardiac malady he encountered among soldiers; he also recognized that the disorder existed in the civilian population as well.

Symptoms included palpitation of varying severity and frequency, with attacks lasting for several hours, attended by increased pain in the cardiac region, accompanied by a great deal of distress. "The 'seizures' were . . . most readily excited by exertion, and might be then so violent, that the patient would fall to the ground insensible." "The rapid action was often commented on; but a slow, hard beat of the heart was also spoken of." The fits of palpitation were associated with cardiac uneasiness and pain, headache, dizziness of vision, and giddiness.

Pain was an almost constant symptom; often it was the first sign noticed by the patient. It was generally described as occurring in paroxysms, and as sharp and lancinating, tearing or cutting. In the large majority there was a substratum of discomfort or dull heavy pain. Exercise or exertion generally produced an attack of sharp pain, and a fit of palpitation was apt to do the same. "The chief seat of the pain was the lower part of the praecordia, particularly near the apex" . . . and was associated with sensitiveness in the cardiac region.

The apical impulse was quick, and abrupt or jerky. "Sometimes the sounds of the heart are split . . . in (one case) the impulse was very irregular; there were double beats and intermissions, and one of the cardiac sounds — the irregularity made it difficult to determine which, though I think it was the first — was curiously broken, and sounded like the sudden motion of an only slightly elastic or cartilaginous substance."

"Murmurs obscuring or replacing the cardiac sounds are not as a rule present; yet they are met with, and particularly in that form of murmur, systolic, chiefly above the apex . . ."

DaCosta described follow-up with "perfect recovery," "amelioration" with irritable condition of the heart remaining, or disordered function leading to organic disease (hypertrophy).

Lewis described "Soldiers Heart and the Effort Syndrome" in soldiers in World War I (1919). He realized that the "effort syndrome" involved a very mixed group (unrecognized infection, convalescence, a small group with incipient but unrecognized heart disease), but dealt with large groups with constitutional weakness (nervous, physical or both) or those "played out" by exposure and strain. He recommended an attitude of deferred judgment and inquiry when considering cause.

He discussed breathlessness, noted vital capacity to be only a little below normal, and suspected an alteration in the character of the blood (acidosis as produced by CO₂ or lactic acid) as a causative factor. Fatigue was an almost universal complaint, with symptoms those which were found in healthy subjects submitted to strenuous exercise. Chest pain was common, frequently was referred to the left side of the chest (the precordium), described as an ache or soreness, frequently exercise related. Palpitation was a frequent complaint — "rapid and energetic heart action" in the majority, occasionally effort related extrasystoles, and rarely paroxysmal tachycardia or atrial fibrillation. Excessive heart rates were found in most of these subjects, and . . . "the pulse shows an exaggerated reaction to posture." The blood pressure was "rarely more or less than normal."

Describing the signs and symptoms as contrasted with those of heart disease, Lewis commented "it is because these symptoms and signs are largely, in some cases wholly, the exaggerated physiological responses to exercise . . . that I term the whole the 'effort syndrome'."

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Most of the soldiers came from sedentary occupations and a large percentage of the patients was affected by the condition in civil life many years before joining the Army. Many showed defective development; "in some the chest is long and narrow, or flattened and associated with a kyphotic curve; in others there is general under-development . . ."

"... amongst any large group of patients who suffer from the 'effort syndrome' a number is discovered in which nervous manifestations are more or less prominent . . . that the syndrome is produced by a primary defect of the nervous system is not likely. Nevertheless a proportion of the patients whom I include in the group effort syndrome sooner or later acquires the diagnosis of neurasthenia."

Grant3 reported on the five year after-histories of 665 men suffering from the effort syndrome in 1925, following a program initiated by Lewis in 1915. One interesting facet concerned the cardiac findings. In 266 cases the heart was reported as being entirely normal. "An apical systolic murmur was present in 233 cases, in 208 of these the murmur was not conducted; in the remaining 25 cases it was noted as being loud and being conducted to the axilla." However, the incidence of "definite cardiac disease" was no greater in patients with cardiac physical signs than among those whose hearts were entirely normal.

By 1933, Lewis' reflected that "... the syndrome is not peculiarly . . . a soldiers (sic) malady or an athletes (sic) malady; it is one of the commonest chronic afflictions of sedentary town dwellers." He thought that possibly several distinct but similar syndromes were being confused, and realized that "for the moment useful subdivision cannot be affected."

Wood5 considered the matter of DaCosta's syndrome or effort syndrome in 1941, following a year's close personal contact with 300 patients and personal researches. He noted that DaCosta's syndrome was commoner in women, and that the "effort syndrome" in the male soldier became cardiac, respiratory, or some other neurosis in the female civilian. He concluded that DaCosta's syndrome should be regarded as an emotional reactive pattern peculiar to psychopathic personalities and to subjects of almost any form of psychoneurosis. "If this conclusion is correct then effort syndrome cannot be accepted as an adequate diagnosis." He suggested that "if it is thought expedient to draw attention to the incapacity for effort, an example of a proper diagnosis might be: anxiety neurosis (effort intolerance)." In the third edition of his text (1968) Wood6 considered the entire matter under the heading of cardiovascular disturbances associated with psychiatric states.

Cohen and White7 spent many years studying neurocirculatory asthenia and summarized their concepts in 1972. They considered the syndrome neurocirculatory asthenia to include two common illnesses, a mild disorder called neurocirculatory asthenia and a more severe illness, manic-depressive disease.

The neurocirculatory asthenia complex (anxiety neurosis, effort syndrome) included cardiovascular symptoms of palpitation, rapid heart beat, chest pain, as well as fatigue, anxiety and dyspnea. "This disorder runs in families . . . twice as many cases occur in women as compared with men . . . patients were generally slight in body build and not well muscled . . . during walking, there was an abnormal rise of blood lactate; during exhausting work, such as running, patients developed significantly high blood lactate concentration." The historical identification with DaCosta and Lewis was described and discussed.

In another sphere, a modest literature dealing with systolic gallop rhythm and systolic clicks (currently designated as the nonejection systolic click) developed during the 1930s and 1940s as an outgrowth of renewed interest in auscultation mediated through phonocardiography. These sounds had engaged the interest of French clinicians since the early 1800s. These clinical studies8, 10 contained a great deal of interesting information: the sounds were relatively common; they occurred frequently in young subjects without evidence of heart disease; psychoneurosis, "nervous people" and functional nervous disturbance were described, as well as accompanying palpitation and sudden, stabbing precordial pain. Postural auscultatory changes and an overall favorable prognosis were present. The incorrect common denominator in these studies was the conclusion that these sounds arose outside the heart.

During the past decade, along with several close associates, I have been involved in the study of patients with what is currently designated as the mitral valve prolapse syndrome. Following the premise that most diseases have been observed previously, I wondered what form the mitral valve prolapse syndrome assumed in earlier days — what did Osler, Lewis, Levine, White or Wood have to say about this clinical syndrome of the 1960s and 1970s? Osler dealt briefly with "neuroses of the heart" and referred back to DaCosta. As I read DaCosta and then Wood, Lewis, Cohen and White, and the auscultatory literature noted above, certain phrases, patient profiles and descriptive clinical terminology appeared to repeat themselves and are easily recognizable in the subjects and patients with mitral valve prolapse. An historical thread runs through these descriptive treatises, defining a one hundred year cycle from DaCosta to mitral prolapse. Patient complaints, signs and symptoms are little changed, only the interpretation has changed. The patients are the same, the doctor's understanding improves.

Certainly many of the patients and subjects described in the earlier syndromes would now be classified under the heading of the incompletely defined and evolving entity, the mitral valve prolapse syndrome. Since Reid,11 Barlow,12, 13 Criley14 and a multitude of workers set the current cycle in motion, the outpouring of information has been incredible, as yet incomplete, and as might be expected, at times contradictory and controversial.

During the past 15 years, the mitral valve prolapse syndrome has been described in almost every country where clinical investigation is performed. A large group of subjects and patients has been described with symptoms of chest discomfort, fatigue, dyspnea, palpitations, tachycardia, anxiety and neurotic behavior; chest wall deformities and asthenic body habitus; peculiar and changing auscultatory findings, previously considered extracardiac in origin, have been related to exuberant, billowing, ballooning, prolapsing mitral and tricuspid leaflets with histologic myxomatous changes; electrocardiographic ST segment, T-wave changes; abnormal electrocardiographic stress tests; a spectrum of
arrhythmias and conduction defects; angiographic and echocardiographic definition of abnormalities of valve motion, and ventricular contraction patterns; abnormal myocardial lactate production or extraction; familial occurrence, and linkages with more precisely defined hereditable disorders of connective tissue.

The purpose of this communication is merely to establish the link between DaCosta, Lewis, neurocirculatory asthenia, and mitral prolapse, to call attention to the giants on whose shoulders we now stand, to fan the still-warm coals of the earlier studies, and to recall Lewis' challenge to bring attitudes of deferred judgment and critical inquiry to our current investigations.

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Acknowledgment

The author appreciates the wisdom of Richard Leighton, M.D., Joseph M. Ryan, M.D., Mary E. Fontana, M.D., Stephen F. Schaal, M.D., William Molnar, M.D., all of whom have contributed to further understanding of the mitral valve prolapse syndrome.

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Circulation. 1976;53:749-751
doi: 10.1161/01.CIR.53.5.749

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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