Historical Perspectives

Cardiology and Myofascial Trigger Points

Janet G. Travell's Contribution

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In 1963, the prospect of a 2-day lecture demonstration on myofascial trigger points (MTrPs) by Janet G. Travell, the White House physician to President Kennedy, delighted me, a staff flight surgeon at the United States Air Force's School of Aerospace Medicine. She was responding to an invitation by my chief, Larry Lamb, MD, who was the lead cardiologist in the USAF and who thought of Janet as a great pioneer in cardiology and myogenic pain. The only thing I knew about MTrPs was that years earlier my chief at the Space Medicine Laboratory in Alamogordo, New Mexico—Col. John P. Stapp, the fastest man on earth via rocket sled—had identified a trigger point as the cause of an enigmatic shoulder pain in a staff member of the laboratory.

Janet's lectures were a revelation to me. So *this* was the cause of most of my muscular aches and pains and those of my friends, family, and colleagues! Although I preferred research to general clinical practice, such an enigmatic yet common myogenic source of human suffering posed an irresistible challenge. The cause of this myogenic pain was clearly overlooked in medical training and practice, but diagnosable and treatable by an expert.

Her demonstrations were awe-inspiring: a focused medical history, a meticulous history of the onset of pain, a detailed identification of the pain pattern, a demonstration of the painfully restricted range of motion, and a finger placed unerringly on the exquisitely tender trigger point. Her application of spray and stretch to that muscle (using Fluori-Methane) always produced immediate and impressive, if not complete, relief of pain and restored full range of motion. Occasionally, she injected a refractory MTrP with 0.5% procaine.

Two years later, in 1965, I retired from the US Air Force and joined the central office of the Veterans Administration (VA, now Veterans Affairs) as coordinator of research for physical medicine and rehabilitation throughout the 63-hospital system. At least once a month, I went to Washington, DC, from my home base in Houston for a few days, in order to communicate with other research coordinators and administrators. My motor-control laboratory, newly computer-equipped, was located in Houston. One of the perks of this job was the regular opportunity to spend the afternoon talking with Janet in Washington before returning to Houston. For nearly 4 years, much of our time was spent discussing her many pharmacologic and muscle-pain experiments to shed light on the pathophysiology of MTrPs and to determine the most effective treatments. I eagerly collected and studied everything that she had written on MTrPs.

During much of this time, she was still working on her autobiography, *Office Hours: Day and Night.*[1] The typescript of this book is among the treasures of George Washington University's Travell Collection. *Office Hours* is a guide to Janet's pioneering spirit and to essentially all of her other publications. Chapter 14, "My Standard of Comfort Was Raised," is delightfully readable, with much enlightening insight into the MTrP enigmas that she studied and into the way that her brilliant and determined mind worked.

In 1930, Janet's 2nd scientific paper, "Importance of Differences in the Potency of Digitalis in Clinical Practice,"[2] set the tone for her initial focus on cardiology and pharmacology. Her 1935 appointment as instructor in pharmacology at the Cornell University Medical School led to a series of more than a dozen papers that were concerned with the pharmacology of cardiovascular disease. She described vividly to me how, when a medical student asked a pharmacologic question to which medical science did not yet have the answer, she would have the student de-

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sign an experiment to answer the question and then help the student to conduct the experiment in the school's pharmacology laboratory.

Janet's introduction to MTrPs came in the mid-1930s, as she tried to massage the muscles over her own scapula to alleviate the pain from an overworked right arm. She was startled when she touched sore spots that intensified her pain as if she had turned on an electric switch.[1] Then she read a 1936 paper[3] that described similar pain with similar trigger zones in the scapular region following myocardial infarction. She soon realized that patients on 3 services had a similar complaint and findings. At the Sea View Tuberculosis Hospital, she was told that the muscle tension and pain was "reflex from the lung, of course." On the Beth Israel cardiac service, the same complaint "came from the heart." On the general medical service, the pain was dismissed, in the absence of organic disease, as psychosomatic. The pain, of course, was so common because the standard of practice was bed rest and immobility.

In 1940, she had the opportunity to do a study of this phenomenon and its treatment by injecting the MTrPs with 1% procaine. A 1938 paper by Kellgren[4] had indicated that these tender spots that had been so unresponsive to treatment could be eliminated by procaine injection if the clinician hit precisely the right spot. Injection of the reference zone, where the patient complained of pain, was ineffective.

A 1941 paper by Hans Kraus[5] inspired Dr. Travell to induce a disbelieving colleague to spray ethyl chloride on the skin over the painful area of an acute myocardial infarction. To his amazement and Janet's delight, it eliminated the pain. Because of the spray's simplicity and lack of side effects, they started to routinely treat pain of both cardiac and MTrP origin by spraying the skin over the painful region with ethyl chloride. Skeptical cardiologists contended that the patients obtained relief only because the spray was treating a simulated cardiac pain, but there could be no doubt that the spray was effective for MTrPs. Soon Dr. Travell was treating a steady stream of grateful employees, staff members, and patients in her private practice who presented with enigmatic (usually MTrP) pain problems.

During this same decade (the 1940s), Janet continued her work with procaine. Her first paper on myogenic pain, "Pain and Disability of the Shoulder and Arm,"[6] described complete relief in 62% of 58 (13 cardiac and 45 noncardiac) patients and moderate-to-considerable improvement in 37%, following procaine injection. It was published in the *Journal of the American Medical Association* in 1942, and the attention it attracted changed the course of her life's work. This approach of procaine injection of MTrPs in patients suffering from the pain of myocardial infarction convinced her and her colleagues that the treatment could stop both noncardiac pain of muscle origin and true cardiac pain of coronary insufficiency. These new findings were first reported in preliminary form in "Relief of Cardiac Pain by Local Block of Somatic Trigger Areas,"[7] and then definitively in 1948 as "Therapy Directed at the Somatic Component of Cardiac Pain."[8] This last article evoked attention mostly in the form of protest, but it deserved serious consideration. Half a century later, clinical recognition of this common source of cardiac-type pain has largely disappeared.

To resolve the cardiac-pain issue and establish that some of the pain that they were relieving with ethyl chloride did originate in the heart, Dr. Travell and her colleague Seymour Rinzler enlisted the help of Isidor Stein and induced angina with ergonovine, monitoring the effect with electrocardiography (ECG). Ethyl chloride spray, applied briefly to the region that was rendered painful by ergonovine, stopped the pain in that area almost at once—faster than nitroglycerin did. However, ECG changes comparable to a "silent" myocardial infarction persisted. Spraying the pain reference zones before the use of ergonovine prevented the pain from appearing or delayed its onset for several minutes, but did not affect the ECG changes of cardiac ischemia. They published these results in 1954, "Blocking Effect of Ethyl Chloride Spray on Cardiac Pain Induced by Ergonovine."[9]

However, a problem remained. There was no published report on the pharmacologic effect of ergonovine on coronary arteries, and the indirect experimental evidence indicated that it caused coronary artery dilation, not constriction. Could they be sure the pain was coming from the heart?

It occurred to Drs. Travell and Rinzler that possibly ergonovine increased circulation only in normal hearts and decreased it in atherosclerotic hearts. This was a heretical concept because all drug testing of this type had been done on normal animals. After much effort, using rabbits that had become atherosclerotic for another study, Janet and her colleagues confirmed the suspected difference in responses[10] and left no doubt that the spray could relieve pain of cardiac origin, as well as of MTrP origin.

They also saw evidence that the spray could suppress cardiac arrhythmias. On several later occasions, I saw the conversion of atrial fibrillation to normal rhythm when vapocoolant spray was applied over the arrhythmia MTrP on the lower-right anterior chest wall; the same effect could be achieved by trigger point pressure release applied to that MTrP, as described in *The Trigger Point Manual*.[11] For several reasons, it appears likely that many such unexpected influences, to and from MTrPs, depend on modulation of the autonomic nervous system, in addition to

modulation of the sensory nervous system (referred pain).

As fully described in chapters 42 and 43 of *The Trigger Point Manual,*[12] the pain patterns of the pectoralis major and pectoralis minor muscles mimic the pain referral patterns of cardiac ischemia. The early studies by Dr. Travell and colleagues provided convincing experimental evidence that the referred pain of cardiac ischemia and the referred pain of active myofascial MTrPs can be eliminated, or decreased remarkably, by application of vapocoolant spray to the skin over the painful area. Clinical studies showed that the 2 sources of pain are easily mistaken for each other[7,8,13,14] and that persistence of pain for some time after the ischemia of a myocardial infarction should have resolved is likely to be caused by MTrPs.[15] Symptoms of angina in the absence of demonstrable cardiac disease should be considered as likely due to MTrPs.

Viewed in the light of recent research, the effectiveness of the application of vapocoolant spray to the skin in the referred pain zone indicates that the spray's afferent input to the dorsal horn blocks transmission of nociceptive stimuli or inhibits awakened dorsal horn nociceptor pathways responsible for the referred pain.[16,17]

Janet's discussions and mentoring inspired me to try to understand what causes trigger points and to become certified as a physiatrist and clinician who treats patients with myofascial trigger points. In 1970, I was accepted as a VA-paid physician in the physical medicine and rehabilitation residency program at the University of Washington in Seattle and began examining all my patients for MTrPs. I tried to treat them on the basis of my memories of our Washington, DC, discussions, supplemented by an occasional telephone call to Janet for guidance; but I realized only modest success at that time.

Upon graduation in 1974, I was assigned a ward of the rehabilitation medical service in the VA Hospital at Long Beach, California. The hospital's education committee supported a 1-month instructional visit by Janet. She spent every Friday afternoon giving a lecture-demonstration to the hospital staff, and the rest of the week demonstrating to me the diagnosis and treatment of MTrPs on my 23 rehabilitation ward patients.

Janet's father had been a skilled amateur magician who had frequently and dramatically displayed his talents to the neighborhood children. This had made a deep impression on her, and her demonstrations of finding and treating MTrPs were laced with consummate showmanship. Usually, I attended these demonstrations; but when I missed and asked afterward how it went, she replied with a sense of spiritual reverence, "the magic never fails." Then she would describe the subjects' problems, her analysis of what caused the MTrPs, how she then demonstrated what was wrong, and the results of her treatment.

As soon as she returned to Washington, we at the VA Hospital realized that we needed written reminders of what she had taught us. From this grew the 1st volume of the *Trigger Point Manual*,[18] which had its basis partly in my weekly Friday evening telephone calls to Washington. During these calls, Janet regularly included exciting descriptions of what she had learned that week from patients—things that she had not known or had never seen before.

During Janet's periodic visits to California, we tape-recorded her answers to my questions so I could continue to write Volume 1 of *The Trigger Point Manual*. Often, a single question would lead to a train of thought that covered many muscles throughout the body. Transcribing and organizing this material was quite a challenge, but Janet's keen mind and meticulous editing made it happen (Fig. 1). The Friday evening telephone calls also helped in the management of patients who had been referred to my VA myofascial pain clinic.

To Janet, every patient was an appropriate subject for a clinical trial of innovative and likely solutions to what was wrong and how to treat it. She looked under

Fig. 1 *Drs. Janet Travell and David Simons, co-authors of the 2-volume textbook* Myofascial Pain and Dysfunction. The Trigger Point Manual. *Photograph 1978.*

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every physical and medical stone imaginable until she found why that patient had failed to respond to treatment as expected. The answers ranged from relatively short upper arms or leg-length discrepancies to inadequate vitamin intake. Her writings in the Travell Collection ("Six Ways to Make Housework Lighter" is a good example) are full of advice on how not to develop MTrPs; this advice arose from her observations of what her patients had done to activate their MTrPs.

During Janet's visits late in the 1970s, we often discussed the question of what causes MTrPs and began to formulate a hypothesis. In 1981, we published our progress to date.[19] That hypothesis explained how the taut band muscle fibers contracted in the absence of propagated electrical activity, and why stretching the muscle could produce rapid resolution of the tenderness of the nodule and the tautness of the band. The hypothesis focused on excessive calcium release from the sarcoplasmic reticulum as a cause of local muscle fiber contracture. The contracture, in turn, causes local ischemia that limits energy replacement and consumes more adenosine triphosphate (ATP), depleting the energy source. These events leave insufficient ATP for adequate return of calcium from the contractile elements to the sarcoplasmic reticulum by the calcium pump. Stretching the muscle reduces the overlap between actin and myosin, thereby reducing energy demand and breaking the cycle.

Beginning in the mid-1990s, others built on the foundation laid by Janet. The mechanism by which pain is referred from an MTrP to the reference zone had remained problematic. A study in the Heidelberg research laboratory of my neurophysiologist colleague, Siegfried Mense,[17] demonstrated 1 such referral mechanism. The awakening of sleeping dorsal horn nociceptive connections by pain from the same muscle or another muscle activates new receptive fields for pain. This observation fits the now-extensive literature on the reconfiguration of spinal cord activity in response to sustained pain input and is described in a book that summarizes these mechanisms and their clinical application to muscle.[16] In addressing ourselves to the key issue of causation, my colleague (John Hong, MD), my physical therapist wife (Lois), and I conducted electrodiagnostic studies on both rabbits[20] and patients.[21] These showed that electromyographic endplate noise is significantly related to MTrPs. Others[22] reached this same conclusion. The evidence that any endplate noise corresponds to greatly increased numbers of miniature endplate potentials[23] indicates that a core feature of MTrPs appears to be the release of greatly increased numbers of acetylcholine vesicles of the motor nerve terminal. A detailed description of the current understanding of MTrP etiology can be found in either of 2 recent books.[24,25]

It would have pleased Janet tremendously to think that someday cardiology residents would be taught how to identify and treat the MTrPs of pectoral muscles that so commonly contribute to, or cause, pain that is assumed erroneously to be of cardiac origin, or pain that becomes enigmatic when all cardiac tests are normal. One must learn how to find which muscle or muscles need to be palpated, learn what to palpate for, and either develop the skill to treat the pain or find a therapist with that skill. Any muscle with a painfully restricted range of motion and a tender spot that reproduces the patient's pain when compressed likely has a myofascial trigger point.

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