Viscerosomatic interaction induced by myocardial ischemia in conscious dogs

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Gwirtz PA, Dickey J, Vick D, Williams MA, Foresman B. Viscerosomatic interaction induced by myocardial ischemia in conscious dogs. J Appl Physiol 103: 511–517, 2007. First published May 3, 2007; doi:10.1152/japplphysiol.00495.2006.—Studies tested the hypothesis that myocardial ischemia induces increased paraspinal muscular tone localized to the T2–T3 region that can be detected by palpatory means. This is consistent with theories of manual medicine suggesting that disturbances in visceral organ physiology can cause increases in skeletal muscle tone in specific muscle groups. Clinical studies in manual and traditional medicine suggest this phenomenon occurs during episodes of myocardial ischemia and may have diagnostic potential. However, there is little direct evidence of a cardiac-somatic mechanism to explain these findings. Chronically instrumented dogs [12 neurally intact and 3 following selective left ventricular (LV) sympathectomy] were examined before, during, and after myocardial ischemia. Circumflex blood flow (CBF), left ventricular contractile function, electromyographic (EMG) analysis, and blinded manual palpatory assessments (MPA) of tissue over the transverse spinal processes at segments T2–T3 and T11–T12 (control) were performed. Myocardial ischemia was associated with a decrease in myocardial contractile function and an increase in heart rate. MPA revealed increases in muscle tension and texture/firmness during ischemia in the T2–T3 segments on the left, but not on the right or in control segments. EMG demonstrated increased amplitude for the T2–T3 segments. After LV sympathectomy, MPA and EMG evidence of increased muscle tone were absent. In conclusion, myocardial ischemia is associated with significant increased paraspinal muscle tone localized to the left side T2–T3 myotomes in neurally intact dogs. LV sympathectomy eliminates the somatic response, suggesting that sympathetic neural traffic between the heart and somatic musculature may function as the mechanism for the interaction.

Cardiac; coronary; osteopathic; somatic; sympathetic

Manual medicine theory suggests that sympathetic pathways between the heart and the upper dorsal paraspinal muscle groups exist and modify muscle tone in response to changes in the physiology of the heart (5, 8, 22, 45, 46, 52). Consistent with this theory are reports of a correlation between ischemic heart disease and the concomitant occurrence of manual or palpatory changes due to “somatic dysfunction” (i.e., impaired or altered function of related components of the somatic system, including skeletal, arthroidal, and myofascial structures and related vascular, lymphatic, and neural elements) in the tissue over the transverse spinal processes at paraspinal segments of the upper dorsal spine in thoracic regions T2–T5 (6–8, 22, 52). Additional corroborative of selective response patterns has been reported from more traditional clinical studies investigating pain patterns with myocardial ischemia (23, 35, 49) and neuroanatomic studies have suggested that these pain patterns may be mediated through sympathetic neural pathways (10, 11, 33, 41). However, definitive evidence of a pathophysiological mechanism in support of the manual medicine reports is lacking.

The one major premise of these manual medicine theories is that the interactions between organs and the somatic responses are mediated by the sympathetic nervous system. From an organ standpoint, the importance of sympathetic nerves in response to pathophysiological stresses, such as myocardial ischemia, has been described (36, 39, 43) and occurs ~10–20 s after the onset of myocardial ischemia. In addition, increases in skeletal muscle tone also appears to be accompanied by sympathetic nerve activity (14, 18). If correct, then manual palpatory assessments (MPA) of paraspinal tissues should correlate with other determinants of paraspinal muscle activity and might provide noninvasive clues to visceral organ dysfunction. It should be noted that by the way these assessments are undertaken, they assess more than motor or muscle responses and, hence, may include sudomotor responses that were not objectively measured in this study. Also, sudomotor responses in the dorsal region differ between human and canine models. The present study was designed to determine whether myocardial ischemia induces a detectable cardio-somatic response in the paraspinal musculature, which localizes to the thoracic region T2–T3 and whether these changes could be detected by palpatory techniques. A secondary component of the study was to determine whether these responses would no longer be detectable when the sympathetic nerves to the heart were severed. Both components are necessary to provide data in support of these theories.

Materials and Methods

Animal selection, surgical preparation, and methods of measurement. All protocols were approved by the Institutional Animal Care and Use Committee at the University of North Texas Health Science Center and were in keeping with American Association for Accreditation of Laboratory Animal Care and US Department of Agriculture guidelines.

Twelve healthy, mongrel dogs of either sex (weight 25–35 kg) were premedicated with acepromazine (0.03 mg/kg sc), anesthetized with surital (5 mg/kg iv), and the trachea intubated. Anesthesia was maintained with isoflurane gas (1–3%) with equal offset of oxygen (1 liter). Using sterile technique, a thoracotomy was performed through the left fifth intercostal space, and the heart was instrumented. A fluid-filled Tygon catheter (1.27 mm OD) was inserted into the aorta just distal to the aortic arch to monitor aortic pressure. A Transonic transit-time Doppler flow probe (Transonic Systems, Ithaca, NY) was placed around the root of the aorta to measure cardiac output (CO). Left ventricular pressure (LVP) was measured by inserting a Konigs-
berg P-6.5 transducer solid-state micromanometer (Konigsberg Instruments, Pasadena, CA) into the left ventricle. A 10-MHz Doppler ultrasonic flow probe (4 mm ID) was positioned around the circumflex artery for measurement of circumflex blood flow velocity (CFV). A pneumatic occluder was placed around the circumflex artery distal to the flow probe to induce circumflex artery stenosis and myocardial ischemia. A heparin-filled Silastic catheter (0.12 mm ID and 0.6 mm OD) was inserted into the circumflex artery distal to the occluder for intracoronary injection of solutions and measurement of coronary blood pressure. Two pairs of miniature ultrasonic 5-MHz piezoelectric transducers were used to measure regional myocardial segment length (SL) by the ultrasonic transit-time method using an ultrasonic dimension system (model 120, Triton Technology, San Diego, CA) (53). One pair was placed in the midmyocardium in the left anterior descending coronary artery perfusion territory (anterior control region), and a second pair was placed in the circumflex artery perfusion territory (posterior ischemic zone). To examine the role of the sympathetic nervous system in mediating the viscerosomatic reflex, three dogs were subjected to selective left ventricular sympathectomy using topical application of 85% phenol to the heart (21, 43, 44). The posterior region of the left ventricle supplied by the circumflex artery was identified and the arteries entering this region carefully dissected. Umbilical tape wetted with phenol was passed under and wrapped around the vessels and removed. Phenol was applied on the epicardial surface and painted from the base to the apex of the heart in a series of interconnecting lines along the anterior boundary to the apical dimple of the left ventricle. The posterior boundary was painted from the apical dimple to the base. Phenol was also applied to the atrioventricular groove above the sympathetomized region. This procedure has been shown to deplete cardiac tissue catecholamine content by 85-90% and not to cause coronary vascular supersensitivity (20, 21).

After instrumentation was completed, a chest tube was placed in the thoracic cavity to evacuate the pneumothorax and posturgical intrathoracic exudate accumulation. Wires and catheters were tunneled subcutaneously to exit between the scapula. Postoperative analgesics, antibiotics, and antipyretics were given as specified by the veterinarian.

Experimental protocols. Dogs were familiarized with the laboratory setting, personnel, and manipulative assessments daily during the 2-wk recovery from surgery. Experiments were conducted while the dog was lying quietly. Experimental protocols were repeated three times on 3 separate days to improve the power of the proposed studies and to eliminate the possibility that a sham stenosis affected the response to a coronary stenosis and vice versa. The MPA was conducted by the specialists in manipulative medicine. Paraspinal segments affected by the myocardial ischemia were thoracic segments T2–T5 (6, 8, 22, 52); segments T11–T12 were used as controls. These spinal regions were then identified, shaved and cleaned for palpatory assessment. The assessment tool consisted of a standardized segmental examination of the tissue over the transverse spinal processes at the predefined levels (described in detail below). The paraspinal muscles are those located next to the spine. Their purpose is to support the spine and provide for movement of the spine. They also function to provide movement between the vertebrae and the rest of the skeleton. The muscles at the T2–T5 region in the dog include the rhomboids, longissimus thoracis, spinalis thoracis, and semispinalis thoracis. The human equivalent to these muscles are the iliocostalis thoracis, longissimus thoracis, and semispinalis thoracis. The human equivalent to these muscles are the iliocostalis thoracis, longissimus thoracis, and semispinalis thoracis. The primary functions of these erector spinae muscles are back extension and posture control in both the dog and human. It must be recognized that a limitation of the present study is the differences in anatomy and function of these muscles between human and the dog (quadraped).

EMG analysis was performed by placing bilateral surface EMG (tran-cutaneous electrical nerve stimulation) electrodes at T4–T5 and T12–T12 over the major paraspinal muscle groups (~1–2 cm. lateral to the midpoint of the spine). Data were only acceptable if the ambient signal was low and increased with motor activity in a pattern consistent with a typical percutaneous EMG. Percutaneous EMG signals were acquired bilaterally during each data acquisition period. Thirty-second averages of the integrated signal were transmitted to a monitoring system. (Mespec 4001 EMG System, Kuopio, Finland). The analog signal was digitized above 200 Hz and analyzed used customized software to determine the mean power frequency (MPF) and geometric mean frequency (MF) for the signal. An increase in the mean power and a shift to a high frequency was taken as evidence of muscle activation.

Coronary artery stenosis and sham stenosis were conducted in random order. The palpatory specialists were blinded to the protocol being conducted. Each specialist received instruction as to the manner of scoring before each trial. Control data consisting of EMG signal acquisition, MPA, cardiac function, arterial blood pressure, and coronary blood flow were collected. The circumflex coronary artery was then either stenosed to reduce coronary flow by 60% or sham stenosed. After 5 min, data acquisition was repeated and the occlusion released. Data were collected 15 min postrelease. At least 30 min elapsed before another procedure was conducted to allow all measured parameters to return to baseline. At no times during coronary artery stenosis did the dog show signs of discomfort or pain.

To evaluate whether myocardial ischemia induced a cardiac sympathetic-mediated reflex, we examined whether the presence of myocardial ischemia induces an increase in a sympathetic α1-adrenergic vasoconstrictor tone in the coronary vasculature (25, 32) in five of the dogs. After control measurements were made, the circumflex coronary artery was stenosed as described above. After 3 min, the selective α1-adrenergic antagonist, prazosin was injected (0.5 mg ic) (25, 32).

MPA. The MPA was derived from standard techniques taught by manual medicine specialists (26) and through agreement with the manual medicine specialists. Each MPA consisted of a segmental examination of the thoracic spine at two predefined levels. This was accomplished by sliding the fingers over the transverse processes and applying a compressive force assessing the response of the superficial and deep paraspinal tissues and the movement of the transverse process. Three components were assessed and recorded: 1) symmetry of movement of the transverse processes, 2) mobility of the spinal segments; and 3) soft tissue quality (skin, subcutaneous tissue, and paraspinal muscles). Asymmetry of the vertebral body movement was evaluated by testing the rotation of the transverse processes by applying alternate equal pressure to the processes of the test vertebrae. Symmetry was defined as equal movement in both direction of the vertebral body. The soft tissue component was evaluated by palpation of the tissues overlying the receptive field. Normal tissue is pliable, relatively uniform, and nontender. In the presence of somatic dysfunction, tissues are typically contracted, irregular, and may elicit signs of discomfort by the animal. A MPA rating of “1” indicated that the tissue was soft, pliable, plastic, mobile, and symmetric (uniform) and that no somatic dysfunction is evident. A MPA rating of “5” indicated that the tissue was hard, irregular, not pliable, resistant to induced movement, bilateral, and marked somatic dysfunction is evident. This MPA rating system was developed in collaboration with manual medicine specialists to objectively quantify osteopathic terminology into a numeric rating system. This MPA rating system was reliable in the same clinician on different days, and between clinicians evaluating the same dog on the same and different days.

Data collection. Online variables recorded and analyzed using PowerLab (AD Instruments, Milford, MA) included left ventricular systolic pressure (LVSP) and end-diastolic pressure (LVEDP), maximal rate of left ventricular pressure development (+dp/dtmax), heart rate (HR), mean arterial pressure (MAP), CO, and CFV. Stroke volume (SV) was calculated by dividing CO by HR. End-diastolic segment length (EDL) and end-systolic segment length (ESL) were identified from the SL signal by referring to the rate of pressure development (dp/dt) signal (53) Percent segment length shortening (%SL) was calculated as [(EDL − ESL)/EDL] × 100. EDL was taken at the time left ventricular dp/dt crossed zero. ESL was taken at the time left...
Fig. 1. Characteristic tracings showing the measured variables in a conscious dog at rest (Control), immediately after stenosis of the circumflex coronary artery, 5 min following stenosis, and immediately after release of the coronary stenosis. LV, left ventricular; SL, segment length.
time of minimal segment length with normally shortening segments (53). To convert CFV to volume rate flow, the circumflex artery diameter within the Doppler probe was measured postmortem. Volume rate was calculated as circumflex blood flow (CBF, ml/min) = (π × D² × V)/4, where D is measured diameter (cm), and V is flow velocity (cm/s).

**Statistical analysis.** Cardiovascular data for the three sham occlusions and three coronary artery occlusions were averaged for each dog and counted only once. Data are reported as means ± SE. An analysis of variance for repeated measures was applied; post hoc comparisons were performed using Tukey’s method. Differences between means were considered statistically significant if P < 0.05 (55).

**RESULTS**

Typical tracings depicting the cardiac and coronary vascular responses to sham stenosis and to circumflex artery stenosis are shown in Fig. 1, and all data are summarized in Table 1. Prestenosis values for all measured variables were similar in all control conditions. Compared with the control state, there were no changes in cardiac or coronary function during the sham coronary artery stenosis. A partial occlusion of the circumflex artery reduced CBF by 58 ± 3%. Circumflex artery stenosis caused significant decreases in +dP/dt max (−16 ± 9%; P < 0.05), posterior regional %SL (−29 ± 14%; P < 0.05), and mean CBP (−48 ± 10%; P < 0.05), indicating ischemic dysfunction occurred during the stenosis. This was accompanied by a significant increase in HR by 21 ± 8% (P < 0.05). These changes persisted during the 5 min of coronary artery stenosis. No significant changes in LVSP, LVEDP, MAP, or anterior regional %SL were observed.

MPA revealed that the muscle tension and texture were soft and compliant bilaterally at both the T4–T5 and T11–T12 segments during control conditions (Table 2). During coronary artery stenosis, the paraspinal muscle tissue texture was reported by the physicians as harder, less compliant, and exhibiting a moderate to heavy degree of muscle tension (P < 0.05) in the T4–T5 segments. Correlated with these MPA findings, EMG recordings demonstrated an increased amplitude for the T4–T5 segments vs. the T11–T12 segments. There were significant increases in both MF and MPF only in the T4–T5 region during myocardial ischemia and postrelease period. An increase in the muscle power and a shift to a high frequency was taken as evidence of muscle activation. Comparison of these data with those obtained in human vastus lateralis muscle (30) indicate similar values at rest. In addition, the level of EMG activity obtained in our dogs during coronary stenosis appear to be equivalent to an exercise workload of 20 W in humans.

Analysis of the time course of the EMG activity demonstrated that the majority of the increase in EMG tone during ischemia occurred between 1 and 4 min after initiation of ischemia. The increase in MPA scores correlated with the rise in segmental EMG tone, but the time course of these findings could not be assessed reliably using the present study design.

### Table 1. Cardiovascular responses to coronary artery stenosis

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Sham Stenosis</th>
<th>Control</th>
<th>Coronary Stenosis</th>
<th>15 min Postrelease</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVSP, mmHg</td>
<td>117±12</td>
<td>127±13</td>
<td>120±13</td>
<td>115±7</td>
<td>117±10</td>
</tr>
<tr>
<td>LVEDP, mmHg</td>
<td>6±2</td>
<td>5±0</td>
<td>8±2</td>
<td>9±4</td>
<td>6±3</td>
</tr>
<tr>
<td>+dP/dt max, mmHg/s</td>
<td>1,867±133</td>
<td>1,867±133</td>
<td>1,567±145*</td>
<td>1,852±120†</td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>108±23</td>
<td>108±23</td>
<td>100±20</td>
<td>119±23*</td>
<td>105±19</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>96±7</td>
<td>93±5</td>
<td>93±5</td>
<td>97±5</td>
<td>96±7</td>
</tr>
<tr>
<td>%SL</td>
<td>14.7±3.7</td>
<td>14.7±3.7</td>
<td>14.7±3.7</td>
<td>9.4±4.9*</td>
<td>14.7±4.0†</td>
</tr>
<tr>
<td>CO, l/min</td>
<td>3.9±0.9</td>
<td>3.8±0.2</td>
<td>3.8±0.2</td>
<td>3.4±0.3*</td>
<td>3.9±0.7</td>
</tr>
<tr>
<td>CBF, ml-min⁻¹·g⁻¹</td>
<td>1.28±0.3</td>
<td>1.28±0.3</td>
<td>1.24±0.2</td>
<td>0.52±0.5*</td>
<td>1.28±0.3†</td>
</tr>
<tr>
<td>CBP, mmHg</td>
<td>95±5</td>
<td>88±4</td>
<td>94±7</td>
<td>46±16*</td>
<td>95±51</td>
</tr>
</tbody>
</table>

Values are means ± SE for 9 dogs. LVSP, left ventricular systolic pressure; LVEDP, left ventricular end diastolic pressure; +dP/dt max, maximal rate of left ventricular pressure generation; HR, heart rate; MAP, mean arterial pressure; %SL, percent segment length shortening; CO, cardiac output; CBF, coronary blood flow; CBP, coronary blood pressure. *P < 0.05 stenosis vs. control. †P < 0.05 coronary stenosis vs. 15 min postrelease of the stenosis.

### Table 2. Manual palpatory and EMG assessment during coronary artery stenosis

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Sham</th>
<th>Coronary Stenosis</th>
<th>15 min Postrelease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual palpatory assessment: T2–T5</td>
<td>1.0±0.1</td>
<td>1.0±0.1</td>
<td>3.8±0.7*</td>
<td>3.0±0.9*</td>
</tr>
<tr>
<td></td>
<td>Bilateral soft, compliant, no SD</td>
<td>Bilateral soft, compliant, no SD</td>
<td>Bilateral hard, SD, marked muscle tension</td>
<td>Bilateral hard, SD, some muscle tension</td>
</tr>
<tr>
<td>Manual palpatory assessment: T11–T12</td>
<td>1.3±0.5</td>
<td>1.7±0.6</td>
<td>2.2±0.5*</td>
<td>1.8±0.4†</td>
</tr>
<tr>
<td></td>
<td>Bilateral; soft, no SD</td>
<td>Bilateral; soft, no SD</td>
<td>Moderate SD, more pronounced on left, muscle tension</td>
<td>Mild SD, little muscle tension</td>
</tr>
<tr>
<td>EMG, µV</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T2–T5</td>
<td>14.5±3.7</td>
<td>25.9±7.3*</td>
<td>19.9±8.3*†</td>
<td></td>
</tr>
<tr>
<td>T11–T12</td>
<td>14.9±5.7</td>
<td>16.9±6.0</td>
<td>14.9±6.1</td>
<td></td>
</tr>
<tr>
<td>MF, Hz</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T2–T5</td>
<td>2.75±1.18</td>
<td>13.93±3.39*</td>
<td>9.75±3.54*</td>
<td></td>
</tr>
<tr>
<td>T11–T12</td>
<td>0±0</td>
<td>1.02±1.02</td>
<td>0±0</td>
<td></td>
</tr>
<tr>
<td>MPF, Hz</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T2–T5</td>
<td>11.45±5.62</td>
<td>25.85±8.10*</td>
<td>20.23±8.34*</td>
<td></td>
</tr>
<tr>
<td>T11–T12</td>
<td>4.38±1.40</td>
<td>14.42±8.43</td>
<td>6.68±3.63</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE for 9 dogs. MF, mean frequency; MPF, mean power frequency; SD, somatic dysfunction. *P < 0.05 stenosis vs. control. †P < 0.05 coronary stenosis vs. 15 min postrelease.
The presence of a sympathetic-mediated $\alpha_1$-adrenergic constrictor tone on the coronary vasculature was examined on separate days by intracoronary injection of the selective $\alpha_1$-adrenergic receptor antagonist, prazosin, in five of the dogs. As expected, prazosin had no effect on coronary blood flow during the sham stenosis (Fig. 2). In contrast, $\alpha_1$-blockade during the coronary artery stenosis resulted in a significant increase in CBF by 23 ± 3% ($P < 0.05$), supporting the hypothesis that coronary artery stenosis leads to an ischemia-induced increase in sympathetic nerve activity (cardiocardiac reflex), which induced a coronary vasoconstriction despite the presence of ischemia.

To confirm the role of the cardiac sympathetic nerves mediating a cardiodynamic interaction, studies were conducted in three dogs subjected to selective left ventricular sympathectomy. The cardiac and coronary responses to coronary artery stenosis were similar to those observed in neurally intact dogs. In contrast to neurally intact dogs, however, there was no palpatory or EMG evidence of increased paraspinal muscle tone. MPA revealed the muscle tension and texture to be soft and compliant bilaterally at both the T4–T5 and T11–T12 segments during stenosis (score = 1.25 ± 0.3; EMG = 14.7 ± 3.2 mV during coronary stenosis).

**DISCUSSION**

These studies demonstrated that there are regional changes in somatic muscle activity during myocardial ischemia that may be due to a cardiodynamic interaction mediated by a direct sympathetic mechanism. In addition, these somatic changes can be identified by palpatory assessment of the paraspinal muscles. Reduction in coronary flow resulted in physiologically significant myocardial ischemia, shown by the decrease in regional myocardial contractile function. The presence of a sympathetic reflex induced by myocardial ischemia is demonstrated by the increase in HR and the presence of an $\alpha_1$-adrenergic-mediated coronary constrictor tone during ischemia (27). After induction of a viscerosomatic reflex, there was evidence of tissue changes and increased muscular tone in the thoracic paraspinal region, indicated by the increase in palpatory scores and EMG activity. These data provide physiological evidence for a segmental, sympathetically-mediated viscerosomatic interaction, initiated by acute coronary ischemia, and selectively affecting the T2–T5 paraspinal muscle groups. Whether this is a reflex mechanism, facilitation, or some other form of physiological interdependency remains to be determined by future studies.

The data also suggest that there was a mild increase in the MPA score in the T11–T12 region with ischemia without a concomitant change in the EMG. An assessment bias is unlikely as any bias would likely favor the T2–T5 region. More likely, these findings may suggest that the MPA is being affected by other physiological processes such as sudomotor activity that was not assessed directly as part of the study protocol but is a component of the MPA. Such a finding would favor a facilitation mechanism over that of a reflex mechanism. Future studies are needed to verify and understand this finding.

Osteopathic manipulative medicine and other manual medicine disciplines have been used for over one hundred years to diagnose and treat a variety of medical conditions. More recently, manipulative interventions have been focused on treating musculoskeletal complaints and as an adjunct to conventional therapies, in conditions such as headaches (9, 14, 31) and pneumonia (12, 47, 48). Osteopathic physicians suggest that manual palpatory findings of the neuromuscular system (i.e., “somatic dysfunction”) can be used to identify visceral dysfunction or disease. In this context, somatic dysfunction refers to impaired or altered function of related components of the somatic system, including skeletal, arthroidal, and myofascial structures, and related vascular, lymphatic, and neural elements. Palpatory evidence of somatic dysfunction is observed at spinal segmental sites related to specific vertebrae. These include objective findings of increased local sudomotor activity, increased skin temperature and moisture, skin texture changes, hyperesthesia, increased subcutaneous fluid, increased muscle contraction or muscle tone, and decreased range of motion (5). These changes are localized at the autonomic reference site of the particular visceral organ involved. Spinal segmental reference areas for visceral organs are related to the autonomic nerve supply for various organs. In general, reference sites for thoracic viscera are from T1 to T5, abdominal viscera are from T5 to T10, and pelvic viscera from T10 to T12.

The conventional explanation for the relationship between palpatory findings and visceral disease is that an interrelationship exists between the somatic musculature and visceral organs, mediated by segmental-specific sympathetic reflex connections. The underlying premise is that reflex stimulation alters the somatic musculature in response to visceral disease or dysfunction via a viscerosomatic reflex (13). However, other forms of interaction, such as a facilitation mechanism, could also account for these findings and be equally valid in the clinical setting. Consistent with this premise, investigators (8, 10, 13, 22, 51, 52) have described clinical and physiological relationships between myocardial ischemia and the somatic paraspinal musculature in the upper dorsal region of the spine. However, direct physiological data demonstrating localized (and palpable) somatic musculature changes in response to myocardial ischemia are limited to anecdotal or correlative studies (8, 10, 13, 22, 51, 52).

Viscerosomatic reflexes are proposed to occur when visceral organ dysfunction initiates an afferent sympathetic stimulus to the somatic musculature. Impulses transmitted to the dorsal...
horn of the spinal cord synapse with interconnecting neurons, and then are sent to both the sympathetic and peripheral somatic nervous systems. The resulting efferent signals would cause sensory and motor changes in somatic tissues. Clinical studies have demonstrated a correlation between myocardial ischemia and somatic changes in the paraspinal muscles overlying the thoracic spine that would be consistent with this hypothesis (7, 8, 22, 52). Unfortunately, these studies only included cardiac patients with preexisting and long-standing disease (e.g., myocardial infarction), on medications that may have complicated the interpretation of the results, and possibly having other end-organ disease. Thus there was not an adequate control condition and the investigators could not clearly establish a direct temporal and causal association with ischemia. The present study used healthy, conscious dogs without preexisting coronary artery or cardiac disease, and not on any medications. Ischemic events were created under tightly controlled conditions, and the examiners were blinded to the conditions. Direct measure of ischemia demonstrated that the response of somatic tissues is immediate and reversible and that the viscerosomatic response, activated by the ischemia, involved the sympathetic connections with the heart.

Neuroanatomic studies provide additional data supporting the findings of this study. Sympathetic postganglionic neurons, typically arising in the stellate and cervical ganglia, project to the epicardial surface of the heart (4), and they innervate the atrial ventricular myocardium, nodal tissue, and the larger coronary arteries. Afferent fibers from the heart carry information regarding mechanical distention, deformation of structures, and the metabolic activity of the heart (1, 10, 17). These afferent neural signals are transmitted to the spinal cord via A-δ- and C fibers and connect with both the spinothalamic and spinoreticular tracts (42, 46, 50). Studies suggest that there is a neural reflex connection between chemoreceptor cells in the myocardium and spinal cord activity (especially neurons in the T1-T5 region) that is mediated by sympathetic afferent fibers (11, 15, 41, 50, 52). These afferent fibers enter the spinal cord, bifurcate, and often traverse several spinal segments. The pathways through which the ascending and descending fibers course is in the marginal zone of the dorsal horn, Lissauer’s tract, the middle of the spinal cord dorsal to the central canal and the dorsal columns (16). There may be numerous collaterals over several spinal segments that arise from these branches and contact other neurons within the dorsal horn (16, 54). Thus the stimulation of primary afferent neurons can activate cells in the dorsal horn over several spinal segments. The convergence-projection theory of referred pain proposes that cardiac and somatic afferent fibers have synaptic connections on the same spinothalamic tract cells. Studies by Jou et al. (34) indicate that activation of sympathetic cardiac afferent fibers produce EMG activity in the spinotrapezius muscles similar to the response evoked by chemical stimulation of cardiac afferent fibers. Transsection of the afferent fibers eliminated this response. Thus their data suggest that activation of spinothalamic tract cells can generate spasmlike muscle contractions that could account in part for anginal pain. We propose that this mechanism may also explain how myocardial ischemia results in the occurrence of somatic dysfunction in the thoracic regions T1–T5.

Approximately 10–20 s after the onset of myocardial ischemia, sympathetic afferent fibers increase their firing rate, initiating their participation in cardiac reflexes (15, 37). This also initiates the sensation associated with myocardial ischemia referred to as angina pectoris (19, 24). Angina often manifests as substernal chest pain or the sensation of chest pressure, but it also includes pain in the precordial anterior thoracic, anterior cervical (typically unilateral) left shoulder, left arm, one or both jaws, teeth, and the posterior chest, i.e., “referred pain.” MacKenzie (40) proposed the “convergence-facilitation” theory to explain these phenomena. According to this theory, sensory impulses originating from the heart are transmitted to the spinal cord and facilitate the signals from other structures such as the skin or somatic musculature. Visceral afferent nerves seem to travel the same fascial pathways of the sympathetic autonomic nerves. The visceral afferent nerves report dysfunction of an organ or tissue to the spinal cord segments of related sympathetic innervation of that organ. This visceral input to the spinal cord contributes to the production of facilitated segments in the spinal cord. In the case of the heart, myocardial ischemia will transmit afferent neural signals generated by the heart to the spinal cord, where they connect with both the spinothalamic and the spinoreticular tract cells. The stimulation of primary afferent neurons can activate cells in the dorsal horn over several spinal segments. Sympathetic efferent fibers distributed along a somatic afferent nerve then complete the viscerosomatic connection. This, in turn, allows for the formation of paraspinal somatic dysfunction in segmentally related somatic structures as demonstrated to occur in this study.

The ability of the heart to sense and respond to ischemia is important for controlling myocardial blood flow distribution, limiting the potential for myocardial ischemic damage and putatively for providing protective information. Cardiac receptors continually assess cardiac function returning information along vagal and sympathetic pathways mediated by A-delta- and C fibers (38). Stimulation of cardiopulmonary afferent fibers has been shown to localize to the T1–T5 segments activating the spinothalamic and spinoreticular tract cells (2, 3, 29), apparently the same regions involved with referred pain in cardiac ischemia as suggested by convergence-projection theories (10, 28, 34). Our data suggest that cardiac afferent activity may cause simultaneous activation of deep muscle groups potentially through a reflex pathway or some other form of physiological interdependency that is highly localized.

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REFERENCES

ISCHEMIC CARDIOSOMATIC INTERACTION