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Understanding Chest Pain: What Every Psychologist Should Know

Steven M. Schwartz,[1,3] Peter C. Trask,[1] and Mark W. Ketterer[2]

Chest pain is one of the most frequent presenting complaints in Emergency Rooms and other medical settings. A considerable number of these patients do not have significant coronary artery disease. This led to plausible alternative explanations for these presenting symptoms and these patients tend to have unremarkable cardiac outcomes. Nevertheless, many studies have also documented that symptoms and related disability persist in the face of reassurances about benign cardiac status. Given the implied threat of chest pain (e.g., myocardial infarction) and the presence of chest pain symptoms in other noncardiac conditions (including anxiety and panic), it is not surprising that many of these patients present with considerable emotional distress. Consequently, chest pain symptoms represent diagnostic and treatment dilemmas for physicians and psychologists alike. The extent to which cardiac and noncardiac factors contribute to all forms of chest pain remains unknown. The function of this review is to provide mental health professionals with a primer on relevant clinical issues in chronic chest pain. We examine several common medical and psychiatric causes of chronic chest pain and selectively review (1) the relevant medical and psychiatric diagnostic and treatment considerations for chest pain and (2) the hypothetical biobehavioral mechanisms relevant to psychological intervention, (3) while expanding on existing conceptual models for understanding chest pain, and (4) offering some suggestions for future research.

KEY WORDS: chest pain; panic; angina; cardiovascular disease; self-management.

[1]Behavioral Medicine Program, University of Michigan Health Systems, Ann Arbor, Michigan.
[2]Henry Ford Health Sciences Center, Detroit, Michigan.
[3]Correspondence should be addressed to Steven M. Schwartz, Ph.D., Behavioral Medicine Program/Department of Psychiatry, University of Michigan Medical Center, 475 Market Place, Suite L, Ann Arbor, Michigan 48108.

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INTRODUCTION

Chest pain is a frequent presenting complaint in many clinical settings (Katon, 1990; Kroenke & Mangelsdorff, 1989) and may be indicative of a number of clinically significant conditions including myocardial infarction, embolus, empyema, angina pectoris, gastroesophageal dysfunction, panic, and/or other forms of emotional distress (e.g., Alpert, Mukerji, Sabeti, Russell, & Beitman, 1991; Alexander, Prabhu, Krishnamoorthy, & Halkatti, 1994; Beitman, Basha, Flaker, DeRosear, Mukerji, & Lamberti, 1987; Beitman, Kushner, Basha, Lamberti, Mukerji, & Bartels, 1991a; Beitman, Mukerji, Russell, & Grafing, 1993; Cannon, Camici, & Epstein 1992; Fleet, Dupuis, Marchand, Burelle, & Beitman, 1994; Fleet, Dupuis, Marchand, Burelle, & Beitman, 1997; Katon, Hall & Russo, 1988; Ketterer, Brymer, Rhoads, Kraft, Kenyon, Foley, Lovallo, & Voight, 1996a; Langevin & Castell, 1991; Lynch, Bakal, Whitelaw, & Fung, 1991; Medalie, 1990; Richter, 1991; Richter, Bradley, & Castell, 1989; Roy-Byrne, Schmidt, Cannon, Diem, & Rubinow, 1989). Kroenke and Mangelsdorff (1989) found chest pain to be the most frequent complaint in an ambulatory care setting. About 7–10% of all patients presenting to the emergency room have chest pain (American College of Emergency Physicians, 1995), making this a significant clinical issue. Diagnostic procedures such as exercise stress testing, thallium stress tests, and/or coronary angiography are useful in identifying cases of coronary ischemia. However, it has been reported that 10–30% of those undergoing costly and invasive angiography have normal or non-significantly obstructed coronary arteries (e.g., Dart, Davies, Dalal, Ruttley, & Henderson, 1980; Kemp, Kronmal, Vlietstra, Frye, & The Coronary Artery Surgery Study Participants, 1986; Kemp, Vokonas, Cohn, & Gorlin, 1973; Lavey & Winkle, 1979; Papanicolaou, *et al.,* 1986; Pasternak, Thibault, Savoia, DeSanctis, & Hutter, 1980; Proudfit, Bruschke, & Sones, 1980). Studies have also found an unremarkable cardiovascular course for these nonischemic chest pain cases (e.g., Bass, Wade, Hand, & Jackson, 1983; Beitman, 1992; Cannon, 1991; Lantinga, Sprafkin, McCroskery, Baker, Warner, & Hill, 1988; Ockene, Shay, Alpert, Weiner, & Dalen, 1980; Papanicolaou *et al.,* 1986; Richter, 1992; Swinson & Kuch, 1990; Wielgosz, Flectch, McCants, Haney, & Williams, 1984). Nevertheless, many of these studies also document continued symptoms and disability in patients even in the face of reassurances about their benign cardiovascular status (Bass *et al.,* 1983; Ockene *et al.,* 1980; Papanicolaou *et al.,* 1986; Potts & Bass, 1993; Swinson & Kuch, 1990; Wielgosz *et al.,* 1984). Because many of these cases have primary or secondary psychiatric conditions relevant to chest pain (e.g., Beitman, Mukerji, Lamberti, Schmid, DeRosear, Kushner, Flaker, & Basha, 1989b; Costa, Zonderman & Engel, 1985; Elias,

Robbins, Blow, Rice, & Edgecomb, 1982) and data support a biobehavioral mechanism of causation, psychologists and other mental health professionals are likely to become increasingly involved in the care of these patients.

Identification and management of chest pain in all its forms remains a complicated and underresearched area that demands increased attention. The function of this review is to provide psychologists and other mental health professionals with a concise primer on the relevant clinical issues in chronic chest pain as they are currently understood. We examine several common medical and psychiatric causes of chronic chest pain and selectively review (1) the relevant psychiatric and medical diagnostic and treatment considerations for chest pain and (2) the hypothetical biobehavioral mechanisms relevant to psychological intervention, (3) while building on existing conceptual models for understanding chest pain by offering a spectrum approach to assessment and intervention using a chronic pain self-management model, and (4) offering some suggestions for future research.

CHEST PAIN OF CARDIOVASCULAR ORIGIN

A number of cardiovascular conditions can produce chronic chest pain. Only two (i.e., angina pectoris and microvascular angina) will be reviewed here as most relevant to this literature. Angina pectoris (AP) results from an imbalance between oxygen demand and supply to the heart muscle secondary to significant buildup of atherosclerotic plaque in one or more critical coronary arteries. Prognosis is most serious in cases where there is considerable blockage of the left main coronary artery (because it feeds the primary pumping portion of the heart), or when three or more of the principal arteries are involved. Diagnosis of angina is complicated in that symptoms can be vague and variable. Symptoms are sometimes painful, but are more typically reported as "heavy," "pressured," or "tight" (Pantano, 1990). Angina pain can also be accompanied by dizziness, radiation of pain down the arm, nausea, and/or vomiting. While discomfort is often localized substernally, it can also occur in the back, at the base of the throat, or even the jaw. In most cases pain is induced by physical exertion, but in some cases pain or discomfort occurs at rest. Chest pain occurring at rest may be indicative of coronary artery spasm ("Prinzmetal's angina"), microvascular spasm (Cannon, 1991), or panic (Beitman, 1992).

Diagnostic procedures for angina include exercise stress testing, echocardiogram, and cardiac catheterization. Exercise stress testing generally focuses abnormalities in the ST segment of the electrocardiogram (i.e., that segment that reflects the resting interval between heart beats) (Fisch, 1997; VandenBelt, Ronon, & Bedynek, 1979; Scheidt, 1996). However, most

episodes of chest pain are not accompanied by ST segment changes (Krantz, Hedges, Gabbay, Klein, Falconer, Merz, Gottdiener, Lutz, & Rozanski, 1994). Use of the radioisotope thallium or technetium in stress testing is diagnostically more sensitive and allows for imaging of myocardial perfusion, volume, or wall motion. Identification of fixed or reversible "cold spots" or wall motion abnormalities is indicative of diminished blood flow.

Treatments for AP essentially focus on providing the heart with more oxygen or reducing the heart's oxygen demand. Medications such as nitrates (e.g., nitroglycerine), angiotensin-converting enzyme (ACE) inhibitors, and calcium channel blockers are used as vasodilators, designed to open up narrowed coronary vessels so they better feed the heart muscle. Anticoagulants (e.g., aspirin, Coumadin, Ticlid, Persantine) allow blood to pass more freely through narrowed vessels by reducing blood viscosity. Beta blockers and digoxin are used to reduce the heart's work load by slowing down its activity and reducing its oxygen demand. Antilipidemic agents are also used to slow the rate of obstruction by reducing blood-based lipids (Roberts, 1996). Angioplasty ("percutaneous transluminal coronary angioplasty," PTCA) opens blocked vessels mechanistically by inflating a specialized catheter at the point of occlusion and spreading the plague thinly over the vessel wall. Unfortunately 30–35% of patients redevelop clinically significant blockage within 3–6 months (Scheidt, 1996). In some cases, stents (i.e., wire mesh structures) are inserted into the affected segment of the artery via catheterization to help hold the vessel open and reduce the likelihood of restenosis (Fischman *et al.,* 1994).

Of particular interest to psychologists is the fact that emotional distress can provoke ischemia-induced pain and ischemia in the absence of pain (i.e., silent ischemia). For example, Goldberg *et al.* (1996) recently demonstrated that mental stress alters a variety of cardiac functions (ejection fraction, myocardial ischemia, vascular resistance). Blumenthal *et al.* (1995) demonstrated a unique relationship between laboratory-induced ischemia (i.e., mental stress) and ambulatory ischemia monitored *in vivo.* Most recently, Kamarck, Shiffman, Smithline, Goodie, Paty, Gnys, and Yi-Kuan Jong (1998) demonstrated elevated blood pressure and heart rate during daily stress.

Given that mental events can alter cardiac functioning and are also related to pain perception, psychological variables also bear on clinical outcomes in AP patients (e.g., Allison, Williams, Miller, Patten, Bailey, Squires, & Gau, 1995; Haines, Imeson, & Meade, 1987; Medalie, 1990). Razin (1984) reviewed the psychological and behavioral factors relevant to angina pectoris and the psychotherapeutic literature available at that time. Razin concluded then that there were too few strong empirical studies available to endorse a definitive conclusion on efficacy, but limited reports

led Razin to believe that biofeedback and cognitive/behavioral therapies held the most promise. Since that time a number of additional studies have been published (e.g., Amarosa-Tupler, Tapp, & Carida, 1989; Payne *et al.,* 1994; Turner, Linden, van der Wal, & Schamberger, 1995) examining the effects of relaxation, imagery, and cognitive restructuring on chest pain and support the promise identified by Razin. However, like Razin, a more recent review by Lewin (1997) concluded that there were still too few comprehensive treatment studies to support conclusive efficacy.

Microvascular angina (MVA) is a term offered by Cannon and Epstein (1988) to reflect more accurately the presumed underlying pathophysiology of another form of cardiovascular chest pain (i.e., vasospasm of the coronary microvasculature) (Cannon, 1991; Cannon *et al.,* 1992). Studies of coronary flow reserve (i.e., the ability of the microvasculature to dilate) have indicated limited reserve in some chest pain patients during exercise (Cannon, 1983; Opherk, Zebe, Weihe, Mall, Durr, Gravert, Mehmel, Schwarz, & Kubler, 1981), although the various causal mechanisms (e.g., dysfunctional sympathetic stimulation of the heart, cardiac-specific chest pain sensitivity, or abnormal nociception) are largely hypothetical. While microvascular angina has some of the features of true angina, it is partially differentiated by the experience of chest pain at rest, more severe and prolonged pain, and a poor response to antiischemic medications like nitrates (Cannon, Watson, Rosing, & Epstein, 1983).

GASTROESOPHAGEAL DYSFUNCTION

It is estimated that approximately 35% of individuals who present complaining of chest pain with normal coronary arteries have esophageal abnormalities (DeMeester *et al.,* 1982). Several specific gastroesophageal (GE) disorders have been hypothesized to result in chest pain including (1) diffuse esophageal spasm, (2) nutcracker esophagus, and (3) achalasia. Gastroesophageal chest pain has been hypothesized to result from either stimulation of mechanoreceptors (via spasm or distention) or chemoreceptors (via acid or bile) (Richter *et al.,* 1989). The relationship between spasms and esophageal pain is inconsistent and not necessarily diagnostic, as contractions can be produced during times of stress in normal individuals without report of pain (Robinson, Orr, McCallum, & Nardi, 1987). Likewise, nutcracker esophagus, which is characterized by peristaltic contractions of high amplitude, does not necessarily produce chest pain. Studies have also failed to report a reduction in contractions when chest pain has been relieved (Richter *et al.,* 1987), suggesting that there may be no causal relationship between peristaltic contractions and chest pain (Richter *et al.,*

1989). Reflux of stomach acid or bile salts (whose origins are further down the digestive tract) also presumably produce chest pain, but neither acid nor bile reflux is necessarily temporally related to chest pain (Johnson, Winters, Spurling, Chobanian, & Cattau, 1987; Richter *et al.,* 1989). Nevertheless, several symptoms may suggest pain of esophageal origin: (1) prolonged pain duration (i.e., lasting several hours), (2) pain that does not radiate down the arms, (3) meal-related chest pain, or (4) relief with antacids (Davies, Jones, Rhodes, & Newcombe, 1985). Even these symptoms, however, may be observed in some cardiac patients.

Importantly, while 50% of GE pain patients have normal coronary arteries, by implication then as many as 50% of patients also have demonstrable coronary disease (Katz *et al.,* 1987; Svensson, Stenport, Tibbling, & Wranne, 1978). This suggestion of high comorbid prevalence may complicate attempts to rule out pain due to "true" cardiac factors. In addition, the base rates for gastroesophageal disorders in the general population have not been adequately established, making relational claims about the high prevalence in chest pain patients, cardiac patients, or other subgroups problematic. Nevertheless the high comorbidity rate and possible relationship of GE symptoms to psychological constructs may make psychological treatment relevant regardless of the causal relationship between GE dysfunction and chest pain.

PSYCHIATRIC DISORDERS

Several psychiatric disorders may present with chest pain as the central symptom. Panic is the most common and thoroughly researched and includes chest pain as a possible diagnostic indicator (American Psychiatric Association, 1994). Katon (1984) reports that 89% of panic-disordered patients referred by primary care physicians for a psychiatric consult initially presented with somatic complaints (cardiac, gastrointestinal, and neurologic symptoms, respectively, being the most common). Beitman and colleagues have studied and written extensively on the topic of chest pain and panic (e.g., Beitman, 1992; Beitman *et al.,* 1987, 1989b, 1991b, Beitman, Kushner, Lamberti, & Mukerji, 1990). In one study, 58% of nonischemic chest pain cases met criteria for panic disorder (Beitman *et al.,* 1987). Misdiagnosis of panic as cardiac in origin can result in unnecessary health service utilization and iatrogenic morbidity (Edlund & Swann, 1987; Markowitz, Weissman, Ouellette, Lish, & Klerman, 1989; McCullough, Ayad, O'Neill, & Goldstein, 1998). Recently, Fraenkel, Kindler, and Melmed (1996/1997) found that brief assessment of fear-based cognitions could differentiate panic from ischemic chest pain. However, many of these panic/chest pain

patients may not report accompanying fear and are not demographically different from those who do (Beitman *et al.,* 1990).

Somatoform disorders have not been well studied in terms of nonischemic chest pain, but may theoretically present as such. These patients typically complain of vague and variable symptoms including gastrointestinal, sexual, and pseudoneurologic symptoms and pain (including chest pain, palpitations, and shortness of breath). Somatoform disorders that are most likely to present with chest pain include somatization disorder, pain disorder, and hypochondriasis. Some degree of hypochondriacal concern is present in nearly all these patients and this is an avenue of research worth pursuing.

Importantly, we have effective treatment technologies for panic and somatoform disorders that include psychotherapy and a range of medications. Cognitive/behavioral (CBT) psychotherapy is perhaps the most effective treatment for panic (e.g., Barlow & Craske, 1994; Marshall, Pollack, & Otto, 1997) and is based on the premise that via erroneous learning individuals exhibit anxiety and sympathetic arousal under inappropriate conditions. Pharmacotherapies may also be warranted in the form of anxiolytics (usually a high-potency benzodiazepine) or antidepressants (particularly the polycyclic antidepressants) (Beitman, 1992). Several studies have reported increased efficacy of *in vivo* exposure therapies with antidepressants (Mavissakalian & Michelson, 1986; Telch, Agras, Taylor, Roth, & Gallen, 1985). Importantly, medication may also interfere with CBT (Bouton & Swartzentruber, 1991) by negative reinforcement, reducing motivation, impairing learning, lowering perceived control, or increasing fear upon removal of the medication (Bouton & Swartzentruber, 1991; Craske & Barlow, 1993; Marshall *et al.,* 1997). In support of the latter, Beitman (1992) reported that up to 80% of panic disorder individuals relapsed within a few months after medication is withdrawn. It has been suggested (Marshall *et al.,* 1997) that the timing and dosage of both psychotherapy and psychopharmacology may influence outcome. This is clearly another area requiring further empirical study.

COMORBIDITY

One aspect of this literature that may account for some of the conceptual confusion and clinical complexity of chest pain symptoms is the comorbidity of various conditions presented here. Probably the most conspicuous and consistent aspect of the comorbidity literature is the presence of emotional distress across all forms of chest pain. Studies have estimated that 33–46% of nonischemic chest pain patients will meet diagnostic criteria for panic disorder (Bass *et al.,* 1983; Beitman *et al.,* 1989b; Katon *et al.,* 1988).

Roy-Byrne *et al.* (1989) found a 40% incidence rate of comorbid panic disorder in patients diagnosed with MVA. Their MVA subjects also developed panic symptoms in response to lactate infusion. While the study sample was small, these data suggest that sympathetic activation may act as a common pathway for both MVA and panic. Clouse and Lustman (1983) reported that psychiatric disorders (e.g., somatization disorders, anxiety disorders, and depression) were identified in 21 of 25 patients with distal esophageal motility abnormalities. Only Ketterer, Kenyon, Foley, Brymer, Rhoads, Kraft, and Lovallo (1996b) and Beitman *et al.* (1987) have examined specifically depressive symptomatology in nonischemic chest pain patients, but found these patients may also be vulnerable to depression. What is needed is a conceptual framework that bridges these diverse conditions and aids psychologists in assessing and treating the psychological and behavioral components of this clinical conundrum.

A COGNITIVE/BEHAVIORAL-PAIN SELF-MANAGEMENT MODEL

Eifert (1992) and Salkovskis and colleagues (Clark, Salkvoskis, Ost, Breithholtz, Koehler, Wrestling, Jeavons, & Gelder, 1997; Salkovskis, 1992; Warwick & Salkovskis, 1990) have provided well-considered cognitive-behavioral conceptualizations for chest pain of nonischemic origin. Eifert (1992) refers to the condition as "cardiophobia." In cardiophobics a positive feedback system develops whereby sympathetically driven cardiac symptoms of emotional distress (e.g., tachycardia misinterpreted as angina or a heart attack) become the focus of somatic attention. Related avoidance behaviors develop out of negatively reinforced associations with the end result being repeated avoidance through phobic responding. Salkovskis' theory and data (i.e., Clark *et al.*, 1997) indicate that inappropriate illness behavior results from the cognitive misinterpretation of the somatic symptoms of anxiety, which in turn reinforce somatic focus and mediate pain tolerance/threshold. Data suggest that muscle tension in some cases may be causally related to chest pain, and Lynch *et al.* (1991) found that increased chest wall EMG is associated with both somatic and cognitive symptoms of panic. These data suggest that muscle tension in some cases may be causally related to chest pain.

Somatosensory amplification is theorized to be a core phenomenon in hypochondriasis, and may also pertain to chronic chest pain (Barsky, 1979; Barsky & Wyshak, 1990; Cioffi, 1991; Warwick & Salkovskis, 1990). Somatosensory amplification concerns the tendency to amplify interoceptive cues. Both Efiert and Salkovski also suggest that mislabeling of affective states

may contribute to maintenance of the system. The exact causes of this misinterpretation continue to be a matter of investigation and debate. Misinterpretation may arise from an actual variation in visceral perceptual sensitivity (e.g., nociception) or by way of some higher cortical function. An early study by Tyrer, Lee, and Alexander (1980) found that hypochondriacal and anxious subjects were better able to predict their own heart rate reactivity relative to patients with phobic anxiety when exposed to experimental stress, suggesting increased cardiac focus. Gramling, Clawson, and McDonald (1996) recently reported that female hypochondriacal subjects manifested greater cardiac reactivity and slower returns to resting baseline during experimental stress as compared to controls. There is also support for abnormal nociception (Turiel, Galassi, Glazier, Kaski, & Maseri). This study showed that women with nonischemic chest pain have lower pain thresholds and tolerance when compared to women with demonstrable coronary artery disease. Recently Barsky, Orav, Delamater, Clancy, and Hartley (1998) found that emotionally distressed subjects reported more cardiopulmonary symptoms at the same level of exercise capacity. Individual differences in pain threshold/tolerance or cognitive appraisal in these patients may also account for differential treatment seeking. A cognitive-behavioral model and somatosensory amplification fit neatly together in a diathesis stress model. The essential difference in these conceptualizations lies in the level of CNS processing at which the error occurs. Importantly, erroneous processing may occur at more than one level of processing in an escalating positive feedback system.

Psychological conceptualizations and treatments break down when real risk factors are also present. Specifically, these conceptualizations and related interventions require patients to reinterpret chest pain symptoms in a more benign fashion. This could be potentially dangerous if the symptoms are cardiac in origin. The implicit requirement is that patients must balance Type I/Type II error. Given the potentially life-threatening nature of symptoms, it may be hard to convince patients to accept a Type II error (i.e., interpret deadly somatic symptoms as benign) when real cardiac risk factors are present (e.g., family history of heart disease, obesity, stable angina). We propose a spectrum approach to chest pain that builds on the theories of Eifert (1992), Salkovskis (1992), and Barsky (1979). This spectrum approach assumes that there are multiple physiologic pathways for chest pain (e.g., angina, MVA, chest wall muscle tension). Each represents a necessary and sufficient condition for chest pain and none are fully independent. Bouts of chest pain vary both within patients and between patients in terms of the relative contribution of psychophysiologic variables. For heuristic purposes one might consider angina pectoris as anchoring one endpoint of the spectrum and classic panic disorder as anchoring the other.

Such a conceptualization has implications for treatment. The entire spectrum of chest pain patients are likely to benefit from some form of self-management program where the treatment goals are to minimize the negative consequences of a chronic condition. Such a program deemphasizes the notion of a "cure" and instead assists the patient in developing problem- and emotion-focused strategies for coping and adjusting to the full range of sequela resulting from chest pain while optimizing daily functioning. For example, patients with true angina should interpret their symptoms in terms of feedback relevant to physical self-pacing to avoid the "sawtooth" pattern of activity seen in most chronic pain patients. A pure panic patient would be required to interpret his or her physiology very differently, typically being instructed to tolerate the discomfort through introceptive exposure techniques. Despite preliminary evidence for abnormal nociception in nonischemic chest pain, little pain perception research has been conducted with chest pain. Indeed, interventions nested in pain self-management programs that emphasize improved coping strategies, self-pacing, and functional living goals would seem appropriate for any of the disorders discussed here. Interestingly, conceptual models and comprehensive treatment programs for chronic pain (Hanson & Gerber, 1990; Turk, Meichenbaum, & Genest, 1983) have received considerable clinical and research attention, but have rarely been considered with regard to chest pain.

The efficacy of psychotherapeutic or pharmacologic treatments for chest pain is only beginning to be substantiated and few treatment outcome studies on nonischemic chest pain symptoms have been published (Beitman *et al.*, 1989a; Cannon *et al.*, 1994; Klimes, Mayou, Pearce, Coles, & Fagg, 1990; Levenkron, Goldstein, Adamides, & Greenland, 1985; Pearce, Mayou, & Klimes, 1990). These data are preliminary, but indicate that both cognitive/behavioral and psychopharmacologic intervention can reduce chest pain (frequency and intensity), increase activities of daily living and reduce utilization of medical services. Both Beitman *et al.* (1989a) and Cannon *et al.* (1994) have evaluated pharmacologic interventions (alprazolam and imipramine, respectively) for nonischemic chest pain. The Beitman *et al.* (1989a) study was an unblinded study with a relatively small sample ($N = 20$) that demonstrated improved panic symptoms, but only a "marginally significant" improvement in chest pain frequency or intensity. Cannon *et al.* (1994) compared imipramine to clonidine in a randomized, double-blind, placebo-controlled trial on chest pain and found that imipramine reduced chest pain symptoms regardless of baseline cardiac, esophageal, or psychiatric status. To date, no study has addressed the issues of additive or synergistic effects of medication plus therapy, nor have more economically viable group or brief therapy formats been tested, with one exception.

Swinson *et al.* (1992) demonstrated that early and brief intervention applied in the ER (i.e., one session exposure instruction for panic) reduced both chest pain and other panic symptoms with treatment gains maintained at 6 months when compared to reassurance alone.

SUMMARY

Chest pain symptoms are common across a broad spectrum of health care settings and can be indicative of any number of conditions ranging from relatively benign (e.g., a single isolated panic attack) to life-threatening (e.g., acute myocardial infarction). Misdiagnosis can result in unnecessary and invasive diagnostic procedures, prolonged use of unnecessary medications (e.g., nitrates, calcium channel blockers), continued disability, and unnecessary health services utilization. Because psychological and behavioral variables appear to be common and relevant to the many forms of chest pain, psychologists will play an increasing role in the assessment and treatment of chest pain patients. Therefore, it behooves psychologists to be aware of the complex clinical issues surrounding chest pain symptoms so they make appropriate clinical decisions regarding referral and treatment. Table I summarizes the critical diagnostic symptom patterns that should be considered during a clinical interview when patients present with chest pain as a central symptom. In such cases the relationship to well-established cardiac risk factors (i.e., smoking, hypertension, family history, morbid obesity, diabetes, sedentary lifestyle, hypercholesterolemia) meals, stressful events, and emotional distress should be evaluated.

Several broad treatment goals should be universally considered. Almost all patients would benefit from mastering some form of psychophysiologic self-regulation (i.e., controlled breathing, relaxation, biofeedback, meditation, etc.). Minimally, these interventions should be intended to circumvent episodes of hyperventilation. Cognitive restructuring may also be required. The specific nature of restructuring would depend on patient characteristics, but must keep in mind the Type I/Type II decision these individuals face with every bout of chest pain. The antecedent conditions that trigger bouts of chest pain may aid patients in the discrimination process. Cognitive restructuring may require helping the patient accurately discriminate subtle psychophysiologic perceptions. Biofeedback (perhaps used in combination with exercise tolerance protocols) may be particularly relevant with this discriminative learning process. Patients prone to amplification may benefit from biofeedback that can help them either better control cardiovascular reactivity or provide them with concrete data regarding the benign relationship between their physiologic functioning, the physi-

Table I. Diagnostic Symptom Patterns[a]

	Chest pain	Heavy	Pressured	Tight sensation	Poor response to antiischematic drugs	Pain with exertion	Jaw pain	Back pain	Fear heaving a serious disease	Pain at rest	Throat pain	Dizziness	Nausea	Radiation down arm	Emotional distress	Prolonged pain	Heartburn	Pain after eating/drinking	Relief with antacids	Difficulty swallowing	Tachycardia	Palpitations	Sweating	Trembling	Shortness of breath	Chills	Rapid onset	Fear of future attacks	Other symptoms in different systems
Angina pectoris	X	X	X	X		X	X	X		X	X	X	X	X	?												X	?	
Microvascular angina	X				X	?				X					X	X											X	?	
Acid/bile reflux	X									X	?	?	?				X	X	X										
Nutracker esophagus	X					?									X			X	X										
Diffuse esophageal spasm	X					?												X	X	X									
Achalasia	X					?												X	X										
Panic disorder	X				?				?			X	X		X	?					X	X	X	X	X	X	X	X	
Somatization disorder	X				?			?	X	?					X	?													X
Pain disorder	X				?			X	?	?					X	?													
Hypochondriasis	X				?			?	X	?					X	?													

[a]X, association; ?, potential association.

cal symptoms, and their cognitive/emotional experience. Emotional distress can also be addressed with models for depression (Beck, Rush, Shaw, & Emory, 1979), panic (Barlow & Craske, 1994), or rational emotive therapy (Walen, DiGiuseppe, & Wessler, 1992) used singly or in combination. What appears to be clear is that if appropriate treatment is not available, these patients will continue to experience increased morbidity, disability, and unnecessary medical system utilization (Bass *et al.,* 1983; Lantinga *et al.,* 1988; Ockene *et al.,* 1980; Papanicolaou *et al.,* 1986; Romeo, Rosano, Martuscelli, Lombardo, & Valente, 1993; Swinson & Kuch, 1990; Wielgosz *et al.,* 1984).

Despite a growing literature, much remains unknown about nonischemic forms of chest pain. The issue of comorbidity is one that has contributed to confusion, and differences in symptom cluster warrants further investigation. Better epidemiologic data on the cooccurrence of the various conditions discussed here may have important implications for treatment development, understanding utilization patterns, and identification of common psychophysiologic pathways. For example, research is needed to identify variables that differentiate panic-masked cardiac cases from those that are more appropriately "captured" by mental health services. Little is known about those panic cases that fall outside of mental health catchment or undergo invasive forms of diagnostic testing. Research assessing patterns of utilization in various clinical settings (e.g., emergency rooms, catheterization labs, primary care, mental health) would be very helpful in devising optimal triaging/treatment protocols. Treatment outcome studies have only recently been published. Given that emotional distress (e.g., depression, panic, generalized and specific phobia) is the one consistent aspect of this otherwise heterogeneous population, one dilemma is devising appropriate psychotherapeutic interventions to help patients with both panic and real cardiovascular risk factors better discriminate their symptoms so as to seek treatment appropriately. Understanding the underlying psychophysiology and individual differences of chest pain patients would support the development of cognitive-behavioral and psychophysiologic interventions. The data on somatosensory amplification and abnormal nociception hold promise in this regard. Interestingly, many of these patients present with hypochondrical cognitive and behavioral characteristics, yet this diagnosis has never been thoroughly applied to nonischemic chest pain. An additional treatment challenge will be creating efficient and effective treatments (both psychotherapeutic and psychopharmacologic) that target patients at critical entry points in the health care system. Finally, further research addressing indirect costs and quality-of-life measures in the context of treatment outcome studies of chest pain with panic would also help broaden our understanding of continued disability in these patients.

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