DOWNLOAD PDF STRESS, CATECHOLAMINES, AND CARDIOVASCULAR DISEASE

Chapter 1 : Catecholamines and Heart Disease - CRC Press Book

The book concentrates on evaluating the stress contribution to cardiovascular disease, presumably because the author is fascinated by the role of the sympathoadrenal system in bridging psyche and soma.

Reprint requests and correspondence: See other articles in PMC that cite the published article. Abstract There is an enormous amount of literature on psychological stress and cardiovascular disease. This report reviews conceptual issues in defining stress and then explores the ramifications of stress in terms of the effects of acute versus long-term stressors on cardiac functioning. Examples of acute stressor studies are discussed in terms of disasters earthquakes and in the context of experimental stress physiology studies, which offer a more detailed perspective on underlying physiology. Studies of chronic stressors are discussed in terms of job stress, marital unhappiness, and burden of caregiving. There is nonetheless overwhelming evidence both for the deleterious effects of stress on the heart and for the fact that vulnerability and resilience factors play a role in amplifying or dampening those effects. In cardiology, complaints of stress take on special prominence because the links between brain and heart are so obvious. This report reviews the literature on this vast topic. Because the review is so broad in scope and touches on blood pressure BP , hemodynamic functioning, arrhythmias, and angina, the review is qualitative, written from the vantage point of 30 years of work in the field. Scope of the Review One senses the vastness of this area because a day does not go by without patients or their families asking about stress and heart disease. Newspapers of greater or lesser repute commonly opine on the matter. A search on PubMed reveals approximately 40, citations. Because of page limitations, it is impossible to cite this enormous literature. The author apologizes to colleagues whose valuable work, while uncited in this review, has nonetheless shaped his thinking and had an enormous effect on the field. If one is to make any sense of this area, one needs to define the intellectual boundaries carefully. This report does not discuss the extensive literature relating cardiovascular disease to depression, vital exhaustion, or type A behavior pattern. Similarly, the report does not discuss exercise stress, oxidative stress, or any of the multiple other physical stressors that impact on the heart. The report also excludes the rich literature derived from animal studies. Nonetheless, the terminology is confusing. They usually mean that they have become embroiled in unpleasant challenging new life circumstances, but sometimes they also refer to challenging new circumstances that are not exactly unpleasant but still demand attention e. Just as cardiologists qualitatively rate the extent of heart murmurs, psychologists and psychiatrists have a variety of ratings schemes for evaluating stress. Some researchers feel that the focus should not be on major life stressors but rather the daily hassles in life rush hour traffic, preparing for Joint Commission on Accreditation of Healthcare Organizations visits, new rules on health care reimbursement, and so on. This report will discuss cardiovascular responses to acute major life stressors as well as chronic exposure to continuing stressors. The report will differentiate acute from chronic stress because the psychological and physiological toll may be different in those settings. The chronic stressor studies include both severe continuing adverse stressors wartime as well as chronic low level aggravations hassles. In either event, with chronic stressors, experimental control is vastly more difficult and insight on underlying mechanisms is more tenuous. This distinction between acute and chronic is arbitrary; an acute sorrow can reverberate for years. Acute Stressors There are 2 basic approaches to studying acute stressors: As an exemplar of chance disasters, this report will review what is known about earthquakes and the heart. This report will then review some of the laboratory findings flowing from experiments on reactivity to stressors. Earthquakes Earthquakes are unique disasters because they come with no warning. Widespread disasters cripple communications and transportation infrastructures. Although a 1-day interruption of electricity and water can be coped with, large-scale disasters typically result in multiple days or weeks of turmoil, inconvenience, worry, and loss. Of course, such disasters have an adverse effect on psychiatric morbidity, but what happens to the heart? Extensive international data demonstrate an increased cardiovascular risk after earthquakes. The underlying mechanism for this risk is unclear. Given the emergency conditions surrounding

a massive earthquake, it is difficult to conduct fine-grained experimental or epidemiologic studies. Thus, it is not known how much of the risk is conveyed by the emotional trauma acting alone versus factors like forgetting to take routine medications, living in cramped emergency quarters, facing disrupted sleep, and so on. Given the chaos surrounding massive disasters, it is unlikely that such data would ever be gathered. During the earthquake in Taiwan, 12 patients were being routinely studied with Holter monitoring. These changes were attenuated in patients who were receiving beta-blockers.

Chapter 2 : Psychological Stress and Cardiovascular Disease

Stress Catecholamines And Cardiovascular Disease Glossary linus pauling institute oregon state university, c reactive protein (crp) a protein that is produced in the liver in response to inflammationcrp is a biomarker of.

In addition, workers in repetitive jobs seem to have difficulties unwinding after work, i. This pattern of catecholamine responses is also consistent with the association between low SES and high catecholamine levels reported by Cohen et al. In view of traditional gender differences in responsibility for unpaid work at home Hall, ; Kahn, ; Lundberg et al. Conclusion Catecholamine responses are strongly related to the intensity of mental stress regardless of its emotional valence. This was demonstrated in early experiments by Levi , in which participants were exposed to films with contrasting emotional content, and by Lundberg et al. Thus, elevated catecholamine levels reflect negative stress as well as strong positive emotions. Urinary catecholamines are particularly useful in the study of occupational psychosocial stress as they reflect the mean stress levels for longer periods of time and do not cause pain or discomfort to the participants. In addition, catecholamines are linked to certain health problems such as cardiovascular disorders. Acta Physiologica Scandinavica Catecholamine and cortisol reaction to child birth. International Journal of Behavioral Medicine, 8, Fear, pain and stress hormones during childbirth. Type of occupation and near-future hospitalization for myocardial infarction and some other diagnoses. International Journal of Epidemiology, 14, Their interaction and regulation. Urinary catecholamines in behavioral research in stress. Ann Arbor, Butterworths, pp. American Journal of Physiology Socioeconomic status is associated with stress hormones. Psychosomatic Medicine, 68, Stress responses in male and female engineering students. Journal of Human Stress, 4, Psychophysiological stress responses in postmenopausal women before and after hormonal replacement therapy. Human Neurobiology, 1, Individual and group differences in psychophysiological responses to stress - with emphasis on sympathetic-adrenal medullary and pituitary-adrenal cortical responses. Consistency in cathecolamine and cortisol excretion in males and females. Outline of a multidisciplinary approach. Journal of Human Stress 2: Women, work and health. Stress and opportunities, New York: The sympathetic-adrenal and pituitary-adrenal response to challenge: Biobehavioral bases of coronary heart disease. Sex differences in sympathetic adrenal medullary reactions induced by different stressors. Sex differences in psychoneuroendocrine reactions to examination stress. Psychosomatic Medicine, 40, Stress on and off the job as related to sex and occupational status in white-collar workers. Journal of organizational behavior, 10, Perceptual and Motor Skills Doctoral Dissertation, Karolinska Institute, Stockholm. Integrative Physiological and Behavioral Science, 1: Stress, Health, and the Social Environment. A Sociobiologic Approach to Medicine. Catecholamine measurements in urine with high-performance liquid chromatography with amperometric detection - comparison with an autoanalyser fluorescence method. Journal of Chromatography, , Women, work, and health. Physiology of stress and regeneration in job related cardiovascular illness. Journal of Human Stress, 8, Consistency in physiological stress responses and electromyographic activity during induced stress exposure in women and men. Integrative Physiology and Behavioral Science 39, Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. The urinary output of adrenaline and noradrenaline during pleasant and unpleasant emotional states. Psychosomatic Medicine, 27, Acta Medica Scandinavica, Suppl. II Psychoneuro-endocrinology human stress and coping processes. Scandinavian Journal of Psychology The influence of paid and unpaid work on psychophysiological stress responses of men and women. Journal of Occupational Health Psychology, 1, Stress hormones in health and illness: Psychoneuroendocrinology, 30 10 , Catecholamine and cortisol excretion patterns in three year old children and their parents. Journal of Human Stress, 7, Stress and workload of men and women in high ranking positions. Journal of Occupational Health Psychology, 4, Psychological and physiological strress responses during repetitive work at an assembly line. Hirsute women with elevated androgen levels: Journal of Psychosomatic Obstetrics and Gynaecology, , 2 2 ,

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Chapter 3 : Cardiovascular reactivity, stress, and physical activity

This is a comprehensive, science-based discussion of how stress, via catecholamines such as adrenaline and noradrenaline, can lead to cardiovascular diseases such as high blood pressure, heart attacks, and sudden death.

Webb Find articles by Heather E. Zourdos Find articles by Michael C. Acevedo Find articles by Edmund O. Received Aug 1; Accepted Oct The use, distribution or reproduction in other forums is permitted, provided the original author s or licensor are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms. This article has been cited by other articles in PMC. Abstract Psychological stress has been proposed as a major contributor to the progression of cardiovascular disease CVD. Combined stress psychological and physical can exacerbate these cardiovascular responses, which may partially contribute to the elevated risk of CVD and increased proportionate mortality risks experienced by some occupations e. Studies have supported the benefits of physical activity on physiological and psychological health, including the cardiovascular response to acute stress. Aerobically trained individuals exhibit lower sympathetic nervous system e. In addition, resistance training has been demonstrated to attenuate cardiovascular responses and improve mental health. This review will examine stress-induced cardiovascular reactivity and plausible explanations for how exercise training and physical fitness aerobic and resistance exercise can attenuate cardiovascular responses to stress. This enhanced functionality may facilitate a reduction in the incidence of stroke and myocardial infarction. Finally, this review will also address the interaction of obesity and physical activity on cardiovascular reactivity and CVD. In addition, acute psychological stress is asssociated with factors that explain the development of atherosclerosis; endothelial dysfunction, inflammatory reactivity and oxidative stress. Examinations of acute responses to psychological stress provide insight into the potential mechanisms that may explain the relationship of psychological stress to CVD. Greater understanding can also provide support for considering therapeutic alternatives that may alleviate the ill effects of stress. Important to our understanding of the development of CVD is how the benefits of physical activity in attenuating the cardiovascular stress response enhanced functionality may also support a reduction in the incidence of stroke and myocardial infarction. Finally, in light of the high prevalence of overweight and obesity

Chapter 4 : MacArthur SES & Health Network | Research

Parkinson disease might be a disease of the elderly because of allostatic load, which depends on genetic predispositions, environmental exposures, repeated stress-related catecholamine release.

Coronary heart disease is also much more common in individuals subjected to chronic stress and recent research has focused on how to identify and prevent this growing problem, particularly with respect to job stress. In many instances, we create our own stress that contributes to coronary disease by smoking and other faulty lifestyles or because of dangerous traits like excess anger, hostility, aggressiveness, time urgency, inappropriate competitiveness and preoccupation with work. These are characteristic of Type A coronary prone behavior, now recognized to be as significant a risk factor for heart attacks and coronary events as cigarette consumption, elevated cholesterol and blood pressure. While Type A behavior can also increase the likelihood of these standard risk factors, its strong correlation with coronary heart disease persists even when these influences have been excluded. However, there is considerable confusion about how to diagnose and measure Type A behavior and numerous misconceptions about which components are the most as indicated in the Interview with Dr. Ray Rosenman, one of the co-authors of the Type A behavior concept. The following discussion is designed to clarify these and other aspects of the role of emotions and behavior in heart disease and how this may relate to the explosive increase in job stress. References have also been provided to obtain additional details on items that may be of special interest. Emotions, Behavioral Traits and Heart Disease: Some Historical Highlights The appreciation that different emotions could have powerful influences on the heart and the recognition of some intimate but poorly understood mind-heart connection is hardly new. Aristotle and Virgil actually taught that the heart rather than the brain was the seat of the mind and soul and similar beliefs can be found in ancient Hindu scriptures and other Eastern philosophies. Broken-hearted, heartache, take to heart, eat your heart out, heart of gold, heart of stone, stouthearted, are just a few of the words and phrases we still use that vividly symbolize such beliefs. William Harvey, who discovered that the circulation of the blood around the body through vessels was due to the mechanical action of the heart also recognized that the heart was more than a mere pump. Osler, He later wrote that he could make the presumptive diagnosis of angina based on the appearance, demeanor and mannerisms of the patient in the waiting room and how he entered the consultation room. Osler, In the s, the Menningers suggested that coronary heart patients tended to be very aggressive. Dunbar, Kemple also emphasized fierce ambition and a compulsive striving to achieve power and prestige. Wolf, In Greek mythology, Sisyphus, the king of Corinth, was doomed by the gods to a life of constant struggle by being condemned to roll a huge marble bolder up a hill, which, as soon as it reached the top, always rolled down again. Wolf characterized people who were coronary prone as constantly striving against real but often self-imposed challenges, and even if successful, not being able to relax or enjoy the satisfaction of achievement. Blood cholesterol level, blood clotting time, incidence of arcus senilis and clinical coronary artery disease. Neither of these two cardiologists had any expertise in psychology, which may have been fortuitous, since they had no preconceived notions. What they did have was an unusual combination of curiosity, diagnostic acumen and a bio-psychosocial approach to the patient as a person, rather than someone to be treated in a cookbook fashion based on laboratory tests, symptoms or signs. As noted, psychiatrists and others interested in psychosomatic disorders had previously described certain personality characteristics in heart attack patients. However, it was not possible to prove that these had any causal relationship since such idiosyncrasies could have resulted from the illness rather than vice versa. Friedman and Rosenman were the first to explain why specific behaviors could cause heart attacks and contribute to coronary artery disease. At the time, animal studies had led to the widespread assumption that heart attacks were due to occlusion of a coronary artery by atherosclerotic deposits resulting from elevated blood cholesterol levels. This, in turn, was primarily the consequence of increased fat and cholesterol intake. Support for this was reinforced by research showing that the significant variation in mortality rates from

coronary heart disease in different countries showed a clear correlation with fat consumption. The greater the amount of saturated fat and cholesterol in the average diet the higher the blood cholesterol and death rate from heart disease in that country. However, Friedman and Rosenman could not confirm this close relationship with serum cholesterol and high fat diet in their heart attack patients and looked for other possible contributing factors. They were intrigued by the observation that two-thirds of the heart attacks in the United States occurred in men, while in Mexico the incidence was equal between men and women. The same equal split appeared to exist in southern Italy but not in northern Italy, where the ratio was four men to one woman. Self-imposed standards that are often unrealistically ambitious and pursued in an inflexible fashion. Associated with this are a need to maintain productivity in order to be respected, a sense of guilt while on vacation or relaxing, an unrelenting urge for recognition or power, and a competitive attitude that often creates challenges even when none exist. Certain thought and activity styles characterized by persistent vigilance and impulsiveness, usually resulting in the pursuit of several lines of thought or action simultaneously. Type A persons often nod or mutter agreement or use short bursts of laughter to obliquely indicate to the speaker that the point being made has already been anticipated so that they can take over. Unsatisfactory interpersonal relationships due to the fact that Type As are usually self-centered, poor listeners, often have an attitude of bravado about their own superiority, and are much more easily angered, frustrated, or hostile if their wishes are not respected or their goals are not achieved. Increased muscular activity in the form of gestures, motions, and facial activities such as grimaces, gritting and grinding of the teeth, or tensing jaw muscles. Often there is frequent clenching of the fist or perhaps pounding with a fist to emphasize a point. Fidgeting, tapping the feet, leg shaking, or playing with a pencil in some rhythmic fashion are also common. Irregular or unusual breathing patterns with frequent sighing, produced by inhaling more air than needed while speaking and then releasing it during the middle or end of a sentence for emphasis. It was also noted that coronary prone patients tend to be very competitive and often overly aggressive. They are usually in a hurry and consequently eat, talk, walk and do most other activities at a more rapid pace. How did the Type A Concept Originate? How the Type A coronary prone behavior hypothesis evolved is a fascinating story, especially since it began because of an interest in cholesterol metabolism rather personality characteristics. Our Harold Brunn Institute for Cardiovascular Research building adjoined the hospital and following early hospital rounds we spent full mornings in the research lab and afternoons in the office. By , although fat and cholesterol had long been fed to rabbits to produce vascular lesions, little was known about where plasma cholesterol came from or how it was metabolized. We also noted that this type of vascular damage was quite different from that seen in patients with coronary artery disease. We obtained Public Health Service and other grants to begin animal studies and Mike was able to solve many fundamental aspects of cholesterol metabolism. I was later able to delineate the mechanisms underlying low and high plasma cholesterol respectivelyi in hypothyroidism and hyperthyroidism and what caused elevated lipids in patients with nephrosis. Around , because of our growing interest in cholesterol, we obtained blood samples from private patients at every visit for no-cost accurate analyses at our research lab. We soon realized that that there were surprising fluctuations in their cholesterol levels that were unrelated to diet or weight, and had little relationship to subsequent coronary events. We subsequently recognized and reported serious errors and omissions in papers by Keys and others about the contribution of diet to plasma cholesterol. The prevailing dogma, which still persists, was that coronary heart disease was due to elevated cholesterol, which in turn resulted from increased dietary fat intake. Our own and other data that Keys had ignored in reaching his conclusions did not support this and reinforced our belief that socioeconomic influences played a more important role in the increased incidence of coronary disease as well as gender differences. These chairs also had to be reupholstered far more often than others because the front edges quickly became worn out. They looked at their watches frequently and acted impatient when they had to wait, usually sat on the edges of waiting room chairs and tended to leap up when called to be examined. Her astute observations significantly reinforced our own awareness of similar behaviors in our coronary patients, then mainly males, that you summarized so well over two decades ago. Occupational pressures and other

sociocultural stresses headed the list. When Rosenman and Friedman subsequently asked the wives, relatives, friends and co-workers of heart attack patients to list possible contributing factors, they were surprised at how often their assessment similarly ranked job stress right at the top. The cluster of behaviors and activity patterns previously described that also emerged from these sources was far more common in males than female. It was also was evident that the current marked increased incidence of coronary disease had occurred mainly in men without any significant change in their diet, increased prevalence of diabetes, hypertension or other risk factors. Even when combined, the standard Framingham coronary risk factors of smoking, hypertension and cholesterol accounted for only about one third of coronary disease patients in prospective studies. It became increasingly clear that these risk factors were merely markers that might predict coronary events but did not cause them. And, whereas simultaneous presence of two or more risk factors is associated with extremely high risk of coronary disease, such situations only predict a small minority of cases. A broad array of recent research studies point with ever increasing certainty to the position that certain psychological, social and behavior conditions do put persons at higher risk of clinically manifest coronary disease. Southern Europe and the U. After a site visit the grant was approved for two years. The methodology of the Western Collaborative Group Study, including the Structured Interview SI for assessing behavior patterns was described in my first follow-up paper. Rosenman, Friedman, Straus et al. We became good friends many years later through you, your annual Congress and other activities of the American Institute of Stress. The significant contribution of Type A behavior to coronary heart disease CHD was subsequently acknowledged by a committee of authorities assembled by the National Institutes of Health The Review Panel , who noted, The Review Panel accepts the available body of scientific evidence as demonstrating that Type A behavior. This increased risk is greater than that imposed by age, elevated levels of systolic blood pressure, serum cholesterol, and smoking and appears to be of the same order of magnitude as the relative risk associated with the latter three of these other factors [p. One problem was that like stress, Type A meant different things to different people. More importantly, researchers also used different assessment or measurement methods so it is not surprising that they reached conflicting conclusions. What they meant by this were observable traits and characteristics that could be readily detected by others, such as the vocal stylistics, breathing patterns, facial grimaces, body movements, hyperresponsiveness and accelerated pace of activities previously described. In their extensive study of employees of several large Western corporations, Rosenman and colleagues were able to predict susceptibility to coronary disease by behavioral characteristics such as a tense, alert and confident appearance; strong voice, clipped, rapid and emphatic speech, laconic answers; evidences of hostility, aggressiveness and impatience, and frequent sighing during questioning. As they noted, Rosenman, Friedman, Straus et al Before and during the personal interview, the following observations upon each subject were made and recorded by the interviewer. In clinical practice, accurate assessment of Type A behavior requires a structured personal interview by a trained investigator using standardized challenges to elicit these tell tale characteristics. For example, one such challenge might be conducted as follows: The investigator begins the interview by asking the following question in a deliberate and painfully slow, monotonous manner. Smith, two second pause , most people, when they go to work during the week â€" that is, Monday through Friday-, get up early two second pause , â€" say around 6: That is probably because it necessary to provide enough time for them to shower, brush their teeth, two second pause and so forth, get dressed, have something to eat, and then they travel by car, bus or train so they can get to work by a certain time two second pause , which is often between 8: How do you travel to work and what time do you usually get there? A flaming Type A would interrupt almost immediately before the question was finished to quickly explain his usual daily routine. Again, the interviewer is not as interested in the content of the response as much as the manner in which it is conveyed and how the subject acts during the interview with respect to facial expressions, gestures, evidence of impatience, time urgency, and other typical Type A traits. Each of these has a certain value and is rated as to severity to obtain a final assessment. Interviews are videotaped so that several reviewers can carefully review the responses and reach agreement on the significance of each component. These Type A characteristics have

been described in detail to emphasize that this complex behavioral pattern can only be accurately assessed by personal observation of the subject by an investigator who has been trained to elicit and evaluate typical responses. Type A behavior is almost impossible to detect in someone who is very sick, bored, depressed, or frightened, such as in a patient recently hospitalized for a heart attack or some other serious medical condition. Reliable ratings therefore require considerable expertise, making large-scale studies quite time consuming and costly. As a consequence, a variety of questionnaires have been devised to detect such aspects of Type A behavior as competitiveness, ambition, impatience, hostility, preoccupation with work, or a constant sense of time urgency. However self-reports fail to capture the stylistics and psychomotor behaviors that are essential to the construct of Type A and its assessment. Self-report questionnaires were rarely validated by those who used them in so many published Type A studies, which also led to considerable confusion in this field. The most commonly used instrument, the Jenkins Activity Survey, detects three main behavioral syndromes: Jenkins, Rosenman, Zyzanski It should be emphasized in evaluating any self-administered questionnaire that Type A individuals are often unaware of many of their behavioral patterns or will deny them. Contrary to popular opinion, there is no rating scale for Type B behavior or definition other than the relative absence of Type A traits. As our understanding and ability to measure Type A improves, it is possible that certain components such as time urgency, latent hostility, aggressiveness, or authoritarianism may be found to have a greater predictive significance for coronary heart disease. Williams This conclusion is based on responses to the Minnesota Multiphasic Personality Inventory MMPI , a item questionnaire developed in that rapidly became the gold standard for psychological testing of hundreds of thousands of college students and prospective employees.

Chapter 5 : Takotsubo Cardiomyopathy

Stress, Catecholamines, and Cardiovascular Disease Stress is an integral part of life. But what is it, how does it affect the body, and what roles do stress and catecholamines play in acute and chronic manifestations of cardiovascular diseases?

Advanced Search The notion that psychological states can influence physical health is hardly new, and perhaps nowhere has the mind-body connection been better studied than in cardiovascular disease CVD. Recently, large prospective epidemiologic studies and smaller basic science studies have firmly established a connection between CVD and several psychological conditions, including depression, chronic psychological stress, posttraumatic stress disorder PTSD , and anxiety. In addition, numerous clinical trials have been conducted to attempt to prevent or lessen the impact of these conditions on cardiovascular health. For each mental health condition, we first examine the epidemiologic evidence establishing a link with CVD. We then describe studies of potential underlying mechanisms and finally discuss treatment trials and directions for future research. One in 5 patients with coronary artery disease or heart failure is depressed, a prevalence that is at least 3 times greater than in the general population. In contrast with other types of CVD, the association between depression and hypertension has been less clear with some, but not all studies showing a modest association. With respect to behavioral factors, depression has been associated with poor adherence to multiple risk reducing health behaviors including physical activity, smoking, and adherence to cardiovascular medications, 17 , 18 and several studies suggest that these factors mediate, at least in part, the association between depression and poor prognosis. For example, poor adherence to physical activity and anti-inflammatory medications may increase inflammation, which may, in turn, increase depressive symptoms. With respect to hypertension, some studies suggest that depression may increase cardiovascular risk by decreasing nighttime blood pressure dipping. Clinical implications and future research directions Though many pharmacological and behavioral therapies are available to treat depression, we do not know which treatments are best to lower the risk of CVD events and mortality associated with depression. Researchers have conducted numerous randomized controlled trials to test whether enhanced treatment of depressive symptoms with conventional antidepressant therapies such as cognitive behavioral therapy, 25 medications, 26 or combination therapy 27 can decrease both depressive symptoms and CVD events. The vast majority of this work has focused on secondary prevention among patients with established CVD. Thus far, conventional depression treatments have only modestly reduced depressive symptoms in patients with a history of CHD or stroke; however, it is worth noting that the effectiveness of such treatments appears to be of the same order of magnitude as when provided to general medical patients. In addition to preventive treatments, questions remain about whether to actively screen patients with CVD for depression. Some argue for screening given its high prevalence, profound impact on quality of life and prognosis, and the availability of brief screening instruments and effective depression treatments. One innovative approach is to develop a clinical service that screens for and provides enhanced collaborative care for common mental disorders, not just depression, among all patients admitted to hospital cardiac services. One trial of such an intervention showed significant reductions in depressive symptoms 0. For example, anti-inflammatory treatments suggest promise as an approach to reducing depressive symptoms in depressed CVD patients. Indeed multiple qualitative studies demonstrate that patients believe daily stressors are key underlying causes of CVD and CVD risk factors, such as poor diet and sedentary lifestyles. A recently published analysis of active duty US military service members participating in the Millennium Cohort Study was able to examine the effects of trauma over a shorter period. The study compared self-report or medical record diagnosis of new CHD events in service members deployed on combat vs. Those who screened positive for PTSD symptoms had an increased likelihood of incident self-report but not medical record CHD diagnoses. This has provided more convincing evidence as well as shed light on the biological changes and possible mechanisms that may be

leading to CVD events. In a study of Vietnam-era veteran twin pairs, Vaccarino et al. Importantly, the authors also found those with PTSD had decreased myocardial blood flow on cardiac positron emission tomography scans. Another study of veterans without known coronary artery disease found those with PTSD had higher levels of coronary artery calcium, a marker of atherosclerosis, on computed tomography scans. The reasons for the lack of classic anginal symptoms are not known but hypotheses include distinct underlying ischemic mechanisms. For example, coronary microvascular dysfunction has been observed in mental stress-induced ischemia and leads to atypical symptoms, such as fatigue and vague discomfort.

Chapter 6 : Stress, Catecholamines, and Cardiovascular Disease - David S. Goldstein - Google Books

Psychological stress has been proposed as a major contributor to the progression of cardiovascular disease (CVD). Acute mental stress can activate the sympathetic-adrenal-medullary (SAM) axis, eliciting the release of catecholamines (NE and EPI) resulting in the elevation of heart rate (HR) and blood pressure (BP).

Open in new tab Alternatively, coronary microvascular and endothelial dysfunction is another hypothesised mechanism of TTC. One of the major arguments for this mechanism is that electrocardiogram ECG changes are diffuse and not suggestive of a single coronary artery territory and on cardiac catheterisation the majority of patients do not have obstructive coronary artery disease. Additionally, the peak troponin level and the extent of ECG changes in patients with TTC are shown to correlate to the severity of endothelial dysfunction. TTC patients compared with post-MI and post-menopausal control women demonstrate more adverse mental stress PAT scores compared with the other groups, consistent with a persistently abnormal physiological response in TTC patients. The theory is that catecholamine release causes LOVT obstruction thus increasing the mechanical stress on the cardiac apex and subsequently leads to myocardial stunning. In previous studies it is shown that norepinephrine causes an increase in cyclic adenosine monophosphate cAMP -mediated intracellular calcium overload that is responsible for myocyte toxicity. Neurogenic stunned myocardium is another mechanism proposed to cause TTC. The fact that an emotional stressor causes TTC suggests that the brain plays a role in inducing cardiac injury. Patients with subarachnoid haemorrhage are found to have elevated catecholamine levels. These patients, particularly females, demonstrate similar findings as those in TTC such as diffuse ST elevation without obstructive coronary artery disease on cardiac catheterisation, significantly reduced wall motion of the apex, and histological changes consistent with those of catecholamine excess, such as contraction band necrosis. Also, by looking at the musculature, the study focused on the SNS activation of the peripheral vasculature but not the heart itself. However, it does suggest that autonomic dysfunction could play a role in TTC. This can further be supported by the fact that many patients with haemorrhage or ischaemic stroke have high occurrence of TTC, and may serve as an alternative explanation for neurogenic stunning. The most common symptoms upon presentation are anginal chest pain and dyspnea. Syncope can also be a presenting symptom but is less frequently observed. Patients can less commonly present with arrhythmias, sudden cardiac death, asystole or cardiogenic shock. Similarly, the T wave inversions are also transient and resolve within 3â€”4 months. The extent of myocardial oedema correlated with that of peak N-terminal of the prohormone brain natriuretic peptide NT-proBNP levels, a marker of inflammation and NT-proBNP values also remained elevated for 3 months. However, some authors do not recommend beta-blockers due to the concern of unopposed alpha stimulation in the setting of catecholamine excess. If LVOT obstruction is present, then preload and afterload optimisation with the use of beta- blockers is recommended. Propranolol was shown to significantly increase left ventricular ejection fraction LVEF in patients with LVOT obstruction, but not in those without it. If dobutamine is given in the setting of LVOT obstruction, it will worsen the ventricular gradient and subsequently exacerbate hypotension. In some case reports, the use of levosimendan, a non-catecholamine inotrope, is shown to be beneficial in treating patients with cardiogenic shock due to pump failure. Anticoagulation is considered in the initial stages where there is severe LV dysfunction, and especially if thrombus is identified in the LV apex. Patients with recurrent episodes can be continued on beta-blocker therapy in addition to stress management. In the majority of cases, the LV wall motion abnormalities resolve in days to weeks. Systolic function can take longer to normalise and on average resolves over 4â€”10 weeks. If LVOT obstruction is responsible for cardiogenic shock, it is usually accompanied by systolic anterior motion SAM of the mitral valve. One study shows TTC patients to have a significantly higher incidence of any malignancy compared with population-matched MI and orthopaedic patients. TTC affects a disproportionately greater number of post-menopausal women compared with pre-menopausal women and age-matched men. It has been reported that catecholamine stress induces

upregulation of immediate early genes IEGs , certain proto-oncogenes and heat shock proteins that are not shown to be activated during reperfusion after an ischaemic episode. Oestradiol-supplemented ovariectomised rats did not show any significant reduction in LV contraction whereas ovariectomised rats without supplementation with oestradiol showed a significant reduction in LV contraction. A recent report indicates that the female heart depends on glucose as its energy source more than the male heart. Males are not predisposed to TTC despite their relative lack of oestrogen because they are not as dependent on glucose as their preferential cardiac energy substrate. Additionally, oestrogen indirectly increases the production of nitric oxide thus promoting vasodilation. Historically, men were exposed to more physical stressors than women and therefore may be better protected biologically than women. It presents with signs and symptoms of ischaemia and acute left ventricular dysfunction with regional wall motion abnormalities in the setting of no obstructive coronary artery disease. An emotional or physical stressor usually precedes TTC. The syndrome has a good prognosis although a few percentage of patients experience recurrent events. Mechanisms implicated in TTC include multi-vessel coronary spasm, endothelial and coronary microvascular dysfunction and direct catecholamine toxicity. Clinicians should be aware of this syndrome and studies that investigate mechanistic pathways of TTC may help with development of preventive and management strategies. Nat Clin Pract Cardiovasc Med.

Chapter 7 : Acute Emotional Stress and the Heart | Cardiology | JAMA | JAMA Network

The starting point for understanding the roles of stress and catecholamines in cardiovascular disorders is a theory defining stress. This book presents a new homeostatic theory of stress and distress and applies this theory to explain the important roles of endogenous catecholamines--norepinephrine, epinephrine, and dopamine--and other effector.

Chapter 8 : Stress and Heart Disease | The American Institute of Stress

While emotional stress has been implicated in the pathogenesis of cardiovascular disease, controversy still exists as to how exposure to stressful situations can induce such pathological changes. Emotional, environmental and sensory stress are all associated with the activation of both the.

Chapter 9 : catecholamines - Heart Disease - MedHelp

to be inappropriate. Even when multiple studies were Although a great deal of research has looked at stress available for a single emotion (e.g., anxiety), they have and stressors as they relate.