MCFIP - After the findings outlined in the following link are reviewed and considered, it becomes obvious that coordination between cardiology and behavioral health professionals is needed to address the primary cause of "stress" and hypertension; i.e. aldosterone. https://www.mcfip.net/upload/Behavioral%20Health%20-%20Brain%20Chemistry(2).pdf

http://dana.org/Cerebrum/2019/Emotional_Rescue_The_Heart-Brain_Connection/

Wednesday, May 08, 2019

Emotional Rescue: The Heart-Brain Connection

By: Michael Miller, M.D.

About the Author

Michael Miller, M.D., is Professor of Cardiovascular Medicine at the University of Maryland School of Medicine and serves on the American Heart Association Leadership Council for Lifestyle and Cardiometabolic Health. He is past president of the American Society of Preventive Cardiology, serves as consultant for the National Library of Medicine and assisted the US Postal Service in developing the "Heart Health Stamp."Miller earned his medical degree at Rutgers Medical School and completed residency at the University of Cincinnati Hospital and fellowships in metabolism and cardiovascular diseases at the Johns Hopkins Hospital. He has published more than 250 original scientific publications, book chapters and three books. His most recent book is *Heal Your Heart, The Positive Emotions Prescription to Prevent and Reverse Heart Disease* (Random House, 2014). Miller's twitter feed is @mmillermd1

Editor's Note: The silent, often subconscious conversation that is taking place inside us is one of the most vital communications we will ever find ourselves engaged in. It's the dialogue of emotion-based signals between our hearts and our brains, also known as the heart-brain connection. Our author tells us what research has uncovered and some of the keys to a longer, healthier life.

We've known for decades that smoking, hypertension, high cholesterol, and diabetes account for most cardiovascular problems. But it wasn't until publication of the Interheart study (25,000 volunteers spanning 52 countries) that emotional stress was identified as another key risk factor, accounting for about one-third of heart attacks and strokes. Previously, in the 1970s, when volunteers were asked to begin to count to 100 and then to serially subtract seven's in quick succession (in a test of "mental stress"), blood vessels constricted as if they had taken and failed a cardiac stress test. Except in these cases, testing occurred at rest.

In other words, external stressors that are not effectively managed have direct internal implications by placing undue stress on the heart. Fast forward from the 1970s to the present era, and a recent study of more than 135,000 men and women in Sweden that found a history of stress-related disorders, such as post-traumatic stress syndrome, increased the risk of cardiovascular disease by more than 60 percent within just the first year of diagnosis.

Mechanistically, the underlying cause of a heart attack is a sudden rupture of an unstable plaque within a coronary artery. During stressful situations, the "fight-or-flight" response jumps into full gear, releasing biochemical compounds such as adrenaline, which raises heart rate and blood pressure, and signals platelets to release a chemical, neuropeptide Y, that can cause spasm and transient occlusion of the coronary artery.

Another cardiac condition that can result from acute emotional stress is Takotsubo cardiomyopathy, named for the Japanese octopus-trapping pot that the heart comes to resemble. Most commonly occurring after a sudden catastrophic event such as losing a spouse, an outpouring of adrenaline creates a transiently "shocked" state characterized by markedly abnormal contractions in a section of left ventricle and by heart failure. Resolution of the emotional crisis coupled with supportive care generally, but not always, leads to recovery of heart function.

Beyond single, severely stressful events, living day-to-day with stress is clearly associated with increased risk of heart attack and stroke. We have only recently begun to understand the neurochemical pathways that generate atherosclerosis and cardiovascular disease. They include close communication between the central nervous system, heart, adrenal gland, and kidneys involved in the activation and release of stress hormones such as cortisol and heart damaging neuropeptides.

On another level, we have come to appreciate that chronic psychosocial or mental stress accelerates cardiovascular disease by promoting inflammation, oxidative stress, and abnormal function of the endothelium, the protective inner lining of our blood vessels.

Connecting to the Brain's Emotional Coding Center

If we are to understand how to improve emotional health, it would be useful to probe the brain's emotional coding center, the amygdala. As an undergraduate at Rutgers University, I had the opportunity to work with Drs. Arthur Kling and Robert Deutsch, a psychiatrist and a neuroscientist doing seminal research into the role of the amygdala in socialization and emotion. After Kling's team induced frontal lobe lesions in rhesus monkeys and severed connections to the amygdala, their social interactions came to a near halt. Similar behavioral patterns have been reported following amygdalotomy for other emotional behaviors in humans, including pathologic aggression. Loss of socialization skills also occurred after prefrontal lobotomy, as I directly encountered when recording social interactions in patients who had undergone the procedure.

The association between high levels of social connectivity and favorable cardiovascular effects, including better outcomes after stroke, raises the possibility that a larger amygdala may afford cardioprotection. The Leiden Longevity Study supports this concept: large left amygdala volumes were not only associated with a high level of emotional health, but also correlated with familial longevity. By contrast, reduced social interactions caused by panic disorders have been associated with reduced amygdala volumes in the lateral and basal regions believed to process fear and anxiety. These disorders correlate with reduced parasympathetic tone, a known contributor to cardiovascular disease risk.

Amygdala activity has also been suggested to play a role in cardiovascular disease risk prediction. For example, residing in high-paced, crowded, noisy, and polluted cities leads to activation of the perigenual anterior cingulate cortex, a brain region that regulates amygdala activity and response to psychosocial stress. Chronic exposure to stress results in allostatic load that adversely impacts brain plasticity and cardiovascular risk factors, including an exaggerated blood pressure response owing to activation of the perigenual cingulate cortex.

In a study conducted in Boston, increased amygdala activity at rest, assessed by PET/CT imaging, was also associated with blood vessel inflammation and risk of cardiovascular events over the next four years. The authors proposed that emotional stress signals a region of the amygdala to activate the sympathetic nervous system, promoting the production of pro-inflammatory white blood cells that may trigger heart attack, stroke, or sudden death. This study, among the first to demonstrate a direct relationship between emotional stressors and risk of cardiovascular events builds upon prior work identifying a direct association between amygdala reactivity (in response to threatening facial expressions) and increased carotid intima-media thickness, an anatomic biomarker of atherosclerosis and cardiovascular risk predictor.

Does counteracting negative stressors reduce cardiovascular risk? While no clinical outcome trials have been conducted to date, adoption of lifestyle strategies aimed at improving positive emotions seems to improve biomarkers of cardiovascular health, such as inflammation, arterial stiffness, and endothelial function. In my cardiology practice and as elaborated upon below, I recommend that my patients employ these five strategies to reduce day-to-day stressors:

1. Meditation (serotonin activated relaxation practices)
2. Yoga (GABA induced mood stabilization)
3. Laughter (endorphin mediated visual effects)
4. Music (dopamine regulated auditory effects
5. Massages, hugging (oxytocin activated tactile responses)

Relaxation Practices

There are several mechanisms by which relaxation strategies such as these improve biomarkers of cardiovascular risk. The first is improvement in parasympathetic tone, the heart's ability to maintain blood pressure and/or heart rate in the face of daily stressors. (This contrasts with the "fight-or-flight" response described earlier, an adaptive physiological mechanism characterized by increased sympathetic tone with associated rise in blood pressure and heart rate). Examples include the inordinate or "hysterical" strength that arose in a daughter attempting to save her father who was pinned under a car and a mother fighting off a lion that attacked her son.

Such isolated "spring into action" situations have no lasting cardiovascular consequences in otherwise healthy individuals. But regularly occurring stressful situations can result in persistently heightened sympathetic tone. Under these conditions, the heart is chronically stressed by exaggerated blood pressure and heart rate responses that endure after the stressful situation is resolved. A persistent increase in sympathetic tone, moreover, raises the likelihood of inflammation, abnormal heart rhythms, and increased risk of sudden cardiac death.

On the other hand, reduced sympathetic or increased parasympathetic or vagal tone enables the heart to manage stressors, keeping blood pressure and heart rate under better control during stress, and shortening recovery time after activities that raise heart rate (such as aerobic activity).

Relaxation strategies like those described above are among the most effective ways to improve

parasympathetic tone. Their benefits are also indicated by tests using heat mapping to evaluate the expression of genes that promote oxidative stress and inflammation, important biomarkers for cardiovascular disease.

One recent study, for example, found that in a group that had practiced meditation on a regular basis, the expression of pro-inflammatory genes was reduced compared to those who had never mediated. In the second stage of the study, one half of the non-meditating group was randomly assigned to relaxation training sessions incorporating meditation, prayer, and yoga. After two months, genetic expression of pro-inflammatory genes resembled that of long-time meditators. Practicing relaxation also reduced the expression of genes promoting insulin resistance, the forerunner of Type 2 diabetes. The results of this study not only affirmed the importance of brain-heart connections on a molecular level but found that relaxation can have a robust effect in a very short time, supporting the adage “never too late to start.”

Mindfulness meditation , which has become one of the most popular relaxation practices over the past decade, combines heightened, non-judgmental awareness of one’s surroundings and feelings with slow deep breathing exercises. A stress-reduction program based on mindfulness has been associated with improvement in hypertension and depression, while strengthening the immune system and raising activity of telomerase, an enzyme that slows biological aging.

Researchers have also studied the cardiovascular impact of practices that incorporate relaxation and movement. Yoga and Tai Chi, for example, improve balance and coordination to help the elderly prevent falls and fractures, and bolster strength and stabilization. In cardiovascular terms, yoga is associated with reduced systolic blood pressure and cholesterol: a recent meta-analysis of 49 trials found that three sessions of yoga weekly reduced systolic blood pressure as much as low-dose antihypertensive medication. Tai Chi has been shown to help suppress inflammation and depression, both cardiovascular disease risk factors. Finally, yoga may also raise brain levels of γ-aminobutyric acid (GABA), a neurotransmitter involved in mood stabilization and stress reduction and both yoga and meditation practices lead to the release of serotonin, another important neurotransmitter involved in mood regulation.

Comic Relief

While it has long been thought that laughter can induce a sense of well-being through the release of endorphins, its connection to cardiovascular health has only become apparent in recent years. Specifically, the β-endorphins released by a hearty belly laugh bind to receptors on the surface of the vascular endothelium to release nitric oxide, a molecule with multiple cardioprotective properties. Recent studies have, in fact, found the risk of heart attack and stroke is reduced in individuals who laugh on a regular basis, compared to those who never or rarely laugh. Laughter also reduces stiffness and aging of blood vessels, including those in the brain.

A popular way to combine laughter with deep breathing techniques is through laughter yoga. The origins of this practice date back to 1995 when Dr. Madan Kataria, a family physician, assembled a small group in a public park in Mumbai, who met each morning to laugh together through a series of funny expressions and movements that Dr. Kataria devised. Nearly 25 years later, more than 15,000 laughter yoga clubs exist in more than 70 countries worldwide.

A typical session lasts from 30 to 60 minutes, during which a leader engages participants in exercises designed to elicit forced laughter that converts to emotional laughter as the session wears on. One popular exercise is “milkshake or cocktail laughter,” where participants pretend to pour a

glass of milk (or cocktail) into one hand saying “here” then into the other hand repeating “here” and then pretending to drink it or discard it behind their shoulder with repeated laughter.

The benefits of laughter yoga include decreased cortisol levels and systolic blood pressure, as well as improvement in indices of depression and overall life satisfaction. While research in this field remains sparse, the encouraging results from these small-scaled studies support the development of a clinical trial in which laughter therapy is one component of an integrated therapeutic lifestyle designed to reduce cardiovascular events.

Music to Your Ears

A number of studies have demonstrated that listening to joyful music offers cardioprotective and neurobiological effects, including reduced inflammation, blood pressure and heart rate, improved parasympathetic tone, and shortened recovery following surgery. The “frisson effect,” or the feeling of chills down the spine is a physiological consequence related to the release of dopamine in response to listening to or anticipating pleasurable music. A pilot study suggested that focusing on this sensation (i.e., mindful music) may be a useful intervention to speed recovery following stroke.

The Moral Molecule

The hormone and neurotransmitter oxytocin, released from the posterior pituitary during physical encounters such as touching and hugging, can lower blood pressure and heart rate. More surprisingly, research in recent years has demonstrated that the compound has a direct cardioprotective effect. In animal models, administration of oxytocin not only prevents the death of heart tissue that results in heart failure but may also regenerate new cells. In human studies, intranasal oxytocin has been shown to improve parasympathetic tone during a mental stress test and may offer relief in chronic pain; the latter has intriguing cardiovascular implications, because chronic pain is associated with increased risk of death from heart disease and stroke.

More work needs to be done to pinpoint the impact of many of the practices mentioned above. But there is already enough research to conclude that effective management of day-to-day psychosocial stressors is vital to good overall heart and brain health. Beyond good nutrition and regular physical activity, then, consider practicing meditation or yoga on a routine basis. Laugh, listen to music, and hug your favorite people and pets. Such are the keys to a longer, happier life.