MATTERS OF THE Heart

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There is an urgency to extend initiatives for cardiovascular health protection, such as increasing awareness for improved life style, nutritious and healthy food, and promote health wellness programmes to combat heart diseases.

"Matters of the Heart" is designed to provide public health education in these areas.

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COVID-19 AND THE HEART

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Corona virus disease 19 (COVID 19) was first reported in December 2019 from China [1]. Later, the disease spread globally and in March 2020, the World Health Organization (WHO) declared the spread of this virus infection as a pandemic [2]. COVID-19 results from infection by Corona Virus 2 (SARS CoV-2), a new virus strain and is a single stranded RNA virus, similar to SARS-CoV and MERS-CoV viruses, which have been recognized earlier to cause acute respiratory distress syndrome (ARDS). Unlike the agents of SARS and the MERS, the virus that causes Covid -19 is highly infective. A majority of individuals infected could remain without any symptoms. About 5 % of patients can have life threatening complications [3]. While the respiratory symptoms are the most common symptoms encountered, it is being increasingly recognized that cardiovascular complications also contribute to the death of patients with Covid -19. A preexisting cardiovascular problem in an elderly patient confers additional risk and most of the deaths that have occurred have been in this group of patients who also have coexisting co-morbidities such as hypertension, diabetes mellitus and chronic respiratory diseases.

Secretions in the form of droplets from the respiratory tract are the main route of transmission of SARS CoV-2, the infectious virus of Covid -19. Infectivity is high with each patient capable of infecting two to three individuals. Incubation period is around 5 days and the mean period for appearance of symptoms is around 12 days. The most common symptoms are fever (99%), fatigue,cough, loss of appetite for food, muscle pain and loss of smell. The common manifestation is that of a pneumonia. In severe cases, breathlessness can rapidly progress to acute respiratory distress syndrome(ARDS) and respiratory failure, the patient needing ventilatory support. Multiple organs can fail as a result of a cytokine storm (cytokines are substances released from immune cells and are of several types) leading to increased leak from blood vessels and tissue damage. Critical illness occurs in about 5% cases and the death rate as is presently known is 2.3 %.The diagnosis is established by examining the throat swab or the secretions collected from the respiratory tract (bronchoalveolar lavage) and doing the RTPC (reverse transcription polymerase chain reaction)test.

Who are at risk?

Individuals with a preexisting heart disease, diabetes, hypertension, especially in the elderly age group are more vulnerable for Covid-19 infections and have a higher risk for poor clinical outcomes. The other groups are higher risk are those with a low immunological status or severe preexisting lung diseases. There are recent reports of Covid-19 infections in young children and infants as well.While younger patients are relatively spared, age is no barrier for a severe infection.

Cardiovascular manifestations

The virus of COVID -19 predominantly affects the respiratory system. Be that as it may, it is now recognized that the cardiovascular effects of the infection are also serious and life threatening. A significant number of patients have evidences of heart damage in the form of elevated blood levels of cardiac troponins. Those patients who had elevated troponins had the highest death rate indicating a more severe and advanced disease in them. Those with an elevated

troponin level in the blood in the initial phases of the disease could be sorted out for a more intense treatment schedule [4].

What are the ways in which the heart can be affected in COVID-19?

1) Direct injury to the heart resulting in inflammation in the heart (myocarditis)
2) Decreased levels of oxygen in blood and tissues (hypoxemia) and increased oxygen demand
3) Heart damage caused by the immune cells
4) Disease related stress resulting in heart muscle disease (cardiomyopathy)
5) Worsening of a preexisting heart disease, resulting in heart attack (acute coronary syndrome)
6) Heart rhythm disorders
7) Clotting of blood in blood vessels and the clots obstructing blood flow to vital organs (thromboembolism)

Many patients with Covid-19 have in their electrocardiograms (ECG) patterns which resemble those seen in a heart attack (acute myocardial infarction with ST segment elevations). As the current treatment for such patients with ST segment elevated myocardial infarction (STEMI) is primary angioplasty, many patients with COVID-19 underwent angiography. They did not however have in their coronary arteries, occlusive clots characteristic of STEMI. Many other ECG patterns mimicking those of heart diseases have been described in patients with COVID-19. The Chinese group and the American group of doctors differ in their opinions regarding management of STEMI. Chinese physicians prefer a conservative approach of thrombolysis (methods to dissolve the blood clot). Patients with direct heart injury and inflammation of heart muscle have poor heart contraction and lower volumes of blood being ejected from the heart. Such patients are prone to havea condition in which the heart suddenly cannot pump enough blood to meet body's needs(cardiogenic shock) and thus extremely poor outcome.

Whether the irregularities of cardiac rhythm observed in patients with COVID-19 is because of the infection per se or from the drugs administered to them is a matter of debate. The use of hydroxychloroquin and azithromycin which are drugs known to lead to ventricular arrhythmias (a serious type of abnormal heart rhythm) may be occasionally fatal. Both these drugs have been advocated for prevention and also for treatment. Authors of a recent article in The Lancet have opined that there may not be significant benefit with these drugs. These drugs are better avoided by individuals who already have a prolonged QT interval.Another factor adding the risk for death in patients with COVID -19 is the development of blood clots in veins (venous thrombosis) and these clots travelling to lungs and occluding blood vessels in the lung (pulmonary embolism). Several reasons could contribute for the increased incidence of venous thrombo-embolism in patients with COVID -19 and include prolonged bed rest,activation of blood clotting mechanisms by infection as well as some of the drugs such as steroids.

What should patients with heart disease do to protect themselves from COVID-19?

It is now clearly documented that patients with preexisting cardiac diseases belong to the highest risk group of patients when they develop COVID-9. They should be extremely careful and avoid contact with patients with COVID-19, especially carriers who have no symptoms of the disease. As widespread testing for COVID-19 is not available at present, the best way for patients with heart disease is to strictly adhere to isolation, social distancing, use of masks andhandsanitizers and strict observance of personal hygiene with frequent handwashing using soap.

The drugs prescribed by Cardiologists should be regularly taken and the dosage adhered to. Routine consultations with the cardiologists can be through electronic media. The post COVID era is going to have a radical change in the frequency of visits of patients to their physicians. There was some confusion regarding the use of angiotensin converting enzyme inhibitors (ACE inhibitors: Ramipril and Lisiopril, Perindopril) and angiotensin receptor blockers (ARBs: Losartan, Telmisartan andOlmesartan) in patients already taking

them, if they develop COVID-19.These are drugs very commonly used for the treatment of hypertension and heart failure and stopping them could worsen heart failure or loss of control of hypertension. The virus that cause COVID -19 binds to the human angiotensin 2 receptor in the lungs. Drugs which block the angiotensin receptors or the converting enzyme could potentiate viral replication and hence these drugs should be stopped during infection according to some authorities..Professionalsocieties such as the European Society of Cardiology and The American College of Cardiology have stated that these drugs need not be stopped and that they do not pose any additional risk [5]. Regular medications such as anti-platelet drugs, statins and anti-anginal drugs should also be not stopped.

An interesting observation is that while COVID-19 spreads like a wild fire across the globe, the number of medical emergencies such as heart attacks (acute coronary syndromes) and strokes have come down. This has not been confirmed as the data are still being collected by various scientific bodies. Many postulates have been put forth for this phenomenon.

As widespread testing for COVID-19 is not feasible currently, there has been a serious concern for the safety of medical professionals actively involved in looking after the patients because of the high virus load they are likely to receive. Among the high-risk medical professionals Cardiologists are at the top of the list of susceptible population. Medical professionals have also to take specific personal protection and hygienic measures to avoid risk to them. Special guidelines have been issued by various professional societies regarding performance of nonemergency heart procedures.

A significant point is that we do not know the long-term implications of this pandemic on atherosclerotic vascular disease in the population.

Key Messages

1) The pandemic of COVID -19 has really shaken the world because of its high infectivity and associated morbidity and mortality. Even the developed nations have struggled to contain and treat this disease effectively.

2) A patient with preexisting heart disease and other comorbidities such as hypertension and diabetes have the highest risk for death when they have COVID-9.

3) Effective treatment strategies for the disease are still evolving.

4) Patients with heart disease, who are stable in their present status should continue the drugs advised by their Cardiologist and strict adhere to the prescribed dosages.

5) Unless there is an emergency, they should avoid visiting hospitals; telephonic or other media can be used for consultations.

6) The current advice is not to discontinue any of the specific drugs used in treatment of hypertension or heart failure.

7) Most important practical way of avoiding infection is to avoid contact with infected patients and to maintain strict social distancing, personal hygiene, frequent sanitization,liberal use of soap for cleaning and washing hands and wearing masks at all situations except when at home.

8) Cardiologists also should be careful for their personal safety, avoid unnecessary exposures and perform their work as per the guidelines and protocols now available.

9) Post COVID era will have a drastic change in patient-doctor communications as well as treatment protocols and management strategies.

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IMPACT OF COVID-19 ON THE HEART

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The outbreak of a novel coronavirus disease was first reported from Wuhan in the Hubei province of China on 8th December 2019. The disease which later came to be known as COVID-19 rapidly spread across continents and was declared a global pandemic by the World Health Organization (WHO) on 11th March 2020. Hardly six months after the first case was reported, as of 5th June 2020, more than 67.5 lakh people have been affected worldwide and close to 2.35 lakh people have been affected in India. As the health-care systems across the world continue to grapple with an exponential surge in the reported cases, it is critical to understand the prognostic factors associated with morbidity and mortality of this new disease. Though the disease primarily affects the lung, a growing body of evidence suggests that multiple organs, particularly the cardiovascular system is affected by the COVID-19 disease. In the present article, we will look at some of the key evidence from the scientific literature on the implications of the COVID-19 disease on the heart.

Several cardiovascular indications have been reported in patients with COVID-19 disease including heart injury, inflammation of the heart (myocarditis), heart failure and heart rhythm disorders. Studies indicate that around 10% of the COVID-19 hospitalizations are associated with elevated serum troponin levels indicative of damage to the heart muscle cells, with the proportions rising to 25-30% or more when the patients are critically ill or present with existing cardiovascular conditions [1]. However, the exact reasons for heart muscle damage in different cases is currently not clear. Further, reminiscent of other acute viral infections, there have been reports of low-grade heart inflammation in the case of COVID-19 disease [2]. Cardiac muscle studies have found inflammatory cell infiltrates and signs of muscle cell death, indicating myocarditis [3]. Though SARS-CoV-2 has been detected in the macrophage infiltrates in the heart, there is currently no clear evidence on whether the virus directly infects the heart muscle cells (cardiomyocytes). In addition to cardiac injury and myocarditis, heart failure has been reported as a significant outcome of COVID-19 in a study of Chinese subjects, with a strikingly higher prevalence among non-survivors (52%) when compared to survivors (12%) [4]. It is possible that heart failure may develop as a consequence of cardiac injury or acute myocarditis although it is difficult to ascertain this. Besides, a study of 138 subjects from a hospital in Wuhan reported heart rhythm disorders (cardiac arrhythmia) and acute cardiac injury in 16.7% and 7.2% of the patients respectively, with patients who received care in intensive care units being more likely to develop these complications [5]. Collectively, these evidences indicate widespread cardiovascular complications in patients with COVID-19 and also indicates a poor prognosis in patients with these complications.

In addition to being one of the major organs affected by the COVID-19 disease, pre-existing cardiovascular conditions also portends a severe clinical outcome upon contracting COVID-19. In one of the most extensive studies which included 44,672 confirmed cases of COVID-19 reported by the Chinese Center for Disease Control and Prevention, the case-fatality rate (CFR) was found to be markedly higher in patients with pre-existing disease conditions [6]. Notably, the CFR in patients with cardiovascular diseases (CVD)

was strikingly high at 10.5%, the highest among patients with other co-morbidities such as diabetes (7.3%), chronic respiratory disease (6.3%) or hypertension (6.0%), while the overall CFR stood at 2.3% [6]. Similarly, many other smaller studies have also noted a similar trend, suggesting an increased risk of adverse events in CVD patients who contract COVID-19 (reviewed in [7]).

Another topic that has received much attention during the pandemic is the relation between certain blood pressure reducing medications and the severity of COVID-19 disease. The involvement of ACE (angiotensin converting enzyme)-2, a vital component of the renin-angiotensin-aldosterone system (RAAS), as a receptor for the SARS-CoV-2 has led to confusion about the use of RAAS blockers such as ACE inhibitors and angiotensin receptor blockers (ARBs) in hypertensive patients. It must be noted here that ACE-2 is distinct from its close homolog ACE, and ACE inhibitors or ARBs do not target ACE-2 directly. Indeed, ACE-2 acts to counterbalance the effects of ACE by hydrolyzing angiotensin II and reduces blood pressure. It was initially hypothesized that a compensatory increase in ACE2 due to RAAS inhibitors could increase the risk of developing severe COVID-19 disease [8]. However, it is essential to note that there is no clear evidence for upregulation of membrane-bound ACE-2 in human tissues due to RAAS inhibitors [9]. Further, in the absence of any established link between RAAS inhibitors and COVID-19 severity and considering that any destabilization of blood pressure in hypertensive patients because of changes in medication can precipitate the risk of strokes and heart attacks, there is no strong justification for discontinuing anti-hypertensive drugs. Several cardiovascular societies such as the European Society of Hypertension, American College of Cardiology and the European Society of Cardiology have now issued guidelines advising against discontinuing anti-hypertensive drugs in the wake of the pandemic (summarized in [10]). Finally, it has even been proposed that increased ACE-2 activity might exert a protective effect against cardiac or lung injury, as it acts to reduce the angiotensin II levels [11].

While the studies and the data discussed here provide vital insights into the cardiovascular sequelae following infection as well as

the severity of COVID-19 in patients with pre-existing cardiovascular conditions, it is important to be mindful of the limitations associated with the studies. Firstly, the vast majority of the initial data has come from China, where the disease was first reported. Second, the majority of the studies reported in the literature are based on retrospective and single-center series. Third, many studies report analyses of small groups or even involve a description of a single isolated case. There is therefore a need to analyze larger groups and include data from diverse populations to arrive at a clearer picture. Further, the clinical presentation of COVID-19 is exceptionally diverse, with more than 80% of the patients being asymptomatic or showing only mild symptoms [6]. When combined with the selection bias in testing strategies and reporting, this can lead to important differences in the estimated prevalence of the underlying risk factors. These shortcomings and the confusion surrounding the use of certain medications can be addressed by prospective cohort studies or randomized controlled trials in the future to address specific questions.

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OBESITY IS A RISK FACTOR FOR COVID-19 INFECTION

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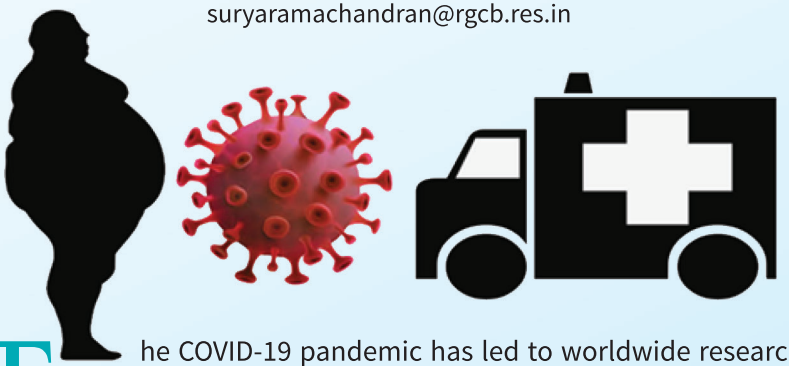

The COVID-19 pandemic has led to worldwide research efforts to identify people at greatest risk of developing critical illness and dying. Initial data pointed towards older individuals being particularly vulnerable, as well as those with diabetes or cardiovascular diseases,hypertension, respiratory or kidney disease. The COVID-19 outbreak has led to an unprecedented health crisis. It is hence crucial to identify individuals who are susceptible to develop severe COVID-19 and could require hospitalisation, especially for admission in the Intensive Care Unit.

Evidence is emerging that obesityrelated conditions seem to

worsen the effect of the virus. As per the World Obesity Organisation, coronavirus infected patients who are obese are significantly at an increased risk of becoming critically ill with the disease. Given the extremely high rates of obesity around the world, it is expected that a high percentage of the population would be at a greater risk of contracting the disease.

As per the National Health Services,body mass index (BMI) below 18.5 means you are underweight, BMI between 18.5 and 24.9 means you are healthy, BMI between 25 and 29.9 range means you are overweight and BMI between 30 and 39.9 range means you are obese.

Studies from Chinese cohorts of patients with COVID-19 have identified several risk factors of severe COVID-19. These include age, heart muscle disease (cardiomyopathy) and obesityrelated complications such as type 2 diabetes and hypertension. Data from the New York City suggest that obesity, as defined by BMI of at least 30 kg/m2, might be a risk factor for ICU admission among patients with COVID-19, especially among those younger than 60 years.

The Lancetpublished on March 27, 2020, a study of 357 patients with COVID-19, who were hospitalised in Lyon University Hospital. Three hundred and forty patients with confirmed and severe COVID-19 were included in the study. Eighty-five (25%) of 340 patients with severe COVID-19 had obesity. The prevalence of obesity was higher in patients with critical COVID-19 than in those with non-critical COVID-19.

These results were compared with those of another study from Lille University Hospital,comprising 124 patients with critical COVID-19 and 306 ICU patients without COVID-19. Among the 306 ICU patients in the Lille population without COVID-19, 79 (26%) had obesity. The prevalence of obesity was higher in patients with critical COVID-19 than in ICU patients without COVID-19. In this study from France, the risk for invasive mechanical ventilation in patients with COVID-19 infection admitted to Intensive Treatment Unit was seven-fold higher for those with BMI >35 compared with normal BMI.

Among individuals with COVID-19 aged less than 60 years in

the New York City, those with a BMI between 30-34 Kg/m2 and >35 Kg/m2 were respectively,1.8 times and 3.6 times more likely to be admitted to critical care than individuals with a BMI. These findings have major implications for the clinical care of patients with obesity and COVID-19, and is also of public health interest

Obesity can weaken the immune system and increase inflammation, and thus makes it difficult for the body to fight off the germs. In addition, obesity also results in excess pressure on the lungs, which further increases the risk of coronavirus complications. Obesity is also associated with other complications such as high blood pressure, diabetes, slow metabolism, sleep apnea, heart disease, stroke, kidney disease and osteoarthritis.Severe obesity, defined as BMI of 40 or above, is a greater risk factor for complications from COVID-19. Severe obesity increases the risk for a serious breathing problem called acute respiratory distress syndrome (ARDS), which is a major complication of COVID-19 and necessitating better respiratory support for seriously ill patients. Severely obese individuals can have multiple serious chronic diseases and underlying health conditions that can increase the risk for severe illness from COVID-19.

Obesity or excess ectopic fat deposition may be a risk factor for severe COVID-19 infection, unifying reduction of both protective cardiorespiratory reserve as well as immune dysregulation which may mediate the progression to critical illness and organ failure in patients with COVID-19. Whether obesity is an independent risk factor for susceptibility to infection requires furtherscrutiny. Obesity and excess ectopic fat lead to impairment of insulin resistance and reduced function of beta-cells which produce insulin.It is possible that the integrated regulation of metabolism required for complex cellular interactions for effective host defense, are lost leading to deficits in immunologic functions.

With extreme obesity (e.g. BMI >40Kg/m2), care for individuals admitted to intensive therapy units is often difficult as these patients are more difficult to be managed for imaging, ventilation, nursing and rehabilitation. There is a clear association between obesity and basal inflammatory status characterized by higher circulating Interleukin 6 and C-reactive protein levels. Adipose tissue in obesity

is “pro-inflammatory”, with increased expression of cytokines and particularly adipokines. Obesity impairs adaptive immune responses to influenza virus and this could be the reason for higher risk of COVID-19in the obese. Obese individuals may exhibit greater viral shedding suggesting potential for greater viral exposure.

It is suggested that BMI as a risk factor should be considered as apredictivetool in those most at risk for severe outcomes. People worldwide should be encouraged to improve their lifestyle to lessen their risk both in the current and subsequent waves of COVID-19. In addition to increasing activity levels, there should be a betterfocus on balanced diet and on simpler measures for sustainable life style changes. This is challenging given the current stay-at-home rules, which limit activity levels. Indeed, this pandemic has highlighted that more, not less, must be done to tackle and prevent obesity for the prevention of chronic diseases and associated adverse reactions to viral pandemics.

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YOGIC MEDITATION - DOES IT REALLY MATTER FOR THE HEART?

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Most people today equate Yoga with physical exercise. The reality is that Yoga was never about physical exercise and postures (asanas), instead, it has much more to offer. Traditionally, the purpose of Yoga was to attain a transubstantiated body endowed with all kinds of paranormal capacities. For this, yoga involved several practices such as mindful breathing (pranayama), meditation (dhyana), sensory withdrawal (pratyahara), concentration (dharana), practicing external and internal (yama- niyama) disciplines and attainment of bliss (samadhi)[1]. Hence, physical fitness was never the prime purpose of yoga, but a welcome by-product of yogic practices. Physical postures or "asanas" were primarily intended to make the body comfortable enough to attune to the meditative state or "dhyana". In this era of lifestyle disorder pandemic, let us see if a 5000-years old practice of yogic meditation can offer some good

news for couch potatoes looking forward to a healthy heart!

There are many different styles and approaches to meditation. It can be categorized as primarily focused attention, mindfulness, loving kindness and compassion, or mantra repetition, although there is usually an overlap between the focuses. Most of them involve sitting comfortably with closed eyes and focusing on breath, mental image, sound, sensation, repetition of positive words and kindness to one and all. The goal is to keep your mind focused on the present and away from stressful or distracting thoughts. By calming the mind, the body is also relaxed.

Based on the critical analysis of studies published over the past two decades, ongoing heart care programs have also started to include meditation. Experts have found that meditation supports heart health in many ways [2]:

Positive effect on Heart Rate Variability (HRV): HRV reflects how quickly the heart makes small changes in the time interval between each heartbeat. A 2013 study found that low HRV is associated with a 32% to 45% increased risk of heart attack or stroke among people without cardiovascular disease. The study revealed that five minutes of meditation daily for 10 days increases HRV, which is a sign of a healthier heart.

Modestly lowers blood pressure: An analysis of nine studies indicate that meditation lower systolic blood pressure (highest number in a reading) by 4.7 milligrams of mercury (mm Hg) and diastolic blood pressure (lowest number) by 3.2 mm Hg. This can be promising to those who wish for a drug free option to lower high blood pressure.

Atherosclerosis regression: Atherosclerosis is a condition which leads toheart attack, stroke and death. The studies on hypertensives and older adults (>65 years) indicated a decrease in the coronary intima media thickness (indicator of atherosclerosis) in the meditation group.

Helps prevent recurrence of cardiac events: A few studies suggest that meditation can lead to improvement in exercise duration and

decrease in myocardial ischemia. A study on 201 individuals with documented coronary artery disease revealed a lower recurrence of cardiac events in the meditation group.

Increased lifespan: Follow up studies on older adults (>75 years) and older hypertensive patients subjected to meditation indicated a 100% survival rate among older adults (65% to 87% in control group) and 81% decrease in death rate in hypertensive patients.

Stress reduction and psychological risk factors: Meditation lowered stress related biomarkers (cortisol, salivary amylase, proinflammatory cytokines etc) and influenced proteomic/ genomic regulators of stress response. Decrease in anxiety, anger and depression was also seen in 47 studies. Brain imaging studies have suggested that mediation can alter brain regions (anterior cingulate cortex [ACC] and mid-cingulate cortex), (hippocampus- regulate emotional response), which control emotional regulation and attenuate connectivity between ACC and amygdala to reduce stress. This is one of the reasons why meditation is considered the cheapest anti-depressant in the market!

Brain and heart connection and other risk factors : Meditators have higher gray matter density in lower brainstem regions which control heartbeat, blood flow, breathing and digestion. Fundamental neural mechanisms underlying meditation involves not only central nervous system processing but also involves neural response to cardiac rhythms[3]. This suggests that even heart beat has much to do with meditation!

Other risk factors: Mindfulness meditation has been shown to increase smoking cessation rates and to have a positive effect on decreasing the consequences of metabolic syndrome and balancing insulin resistance.Twenty minutes of listening to a relaxation response instructional CD reduced expression of genes linked to inflammatory response and the stress-related pathway (mechanisms that contribute to metabolic syndrome) and enhanced the expression of genes associated with energy metabolism, mitochondrial function, and insulin secretion.

It appears that “meditators” were not really couch potatoes

doing nothing. There is much happening in this passive activity which science still needs to unfold!

If you wish to give it a try, use this basic meditation exercise suggested by Dr. Herbert Benson from Harvard [4]:

1. Sit quietly and close your eyes. Breathe slowly.
2. Relax all of your muscles, starting with your feet, legs, and thighs. Shrug your shoulders; roll your neck to the left and right.
3. On each "out" breath, say the word "peace."
4. When thoughts come to mind, decide to come back to them later, and repeat the word "peace."
5. Continue the exercise for at least 10 minutes. Repeat them daily.

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WHAT IS THE LIMIT OF STRESS A HUMAN HEART CAN ENDURE ?

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"Size of yourheart is the size of yourfist", is a common usage we hear and readeventhoughthis statement, is not a systematic techniqueto access the size of heart. How much stress this fist sized heart can withstand? According to a report submitted to Clearvue Health, an online repository of practitioners and experts in the field, emotional stress can increase the risk of heart attack by 1.34 times, stroke by 1.75 times and high blood pressure by 2.15

times. They also report that patients taking antidepressants such as Lexapro had a significantly lower risk of heart attack (46% lower in those taking Lexapro).

According to World Health Organization, cardiovascular diseases (CVDs) are the number one cause of mortality globally, taking approximately 17.9 million lives each year among which 85% die because of heart attack and stroke. The role of stress as a risk factor for CVDs cannot hence be over looked. Considering its deleterious effect, not only in the cardiovascular system but to all systemic organs, stress is now considered as a "World-wide epidemic".

Stress is body's natural defence against danger; when the body will have increased levels of certain hormones, as a part of fight-or-flight mechanism. Stress is a beneficial response elevating spirit and surveillance; at the same time, it can also have a negative impact in a person's life by provoking undesirable consequences. Stress in life has a direct relationship with stress on heart and can hasten heart disease and can ultimately lead to a heart attack. The Global Organization for Stress claims that stress is the number one health concern among high school students. The American Institute of Stress reported that in about half of all the Americans, stress level is getting worse instead of getting better. It is not easy to measure stress levels. Its physical, physiological or psychological manifestations can however be measured. Stress levels would vary in different individuals. Despite the fact that numerous techniques have been developed to aid doctors and mental health physicians to measure the degree of stress elements it is difficult to identify how much stress an individual can tolerate.

Anxiety and stress make your heart work harder, by invoking the fight and flight mechanism which tense the body, increase blood pressure and make the heart beat faster. Stress hormones damage the lining of the arteries and contribute to thickening of the arterial wall. In addition, stress releases fatty acids and glucose into the bloodstream which aid deposits in the inner walls of arteries, thus narrowing their lumen and result in decreased blood flow.

Researchers at University of Maryland reported that smoking

cigarettes or consuming excessive caffeine can raise the heart rate by an average of 14 beats per minute, which increase as much as by 38 beats per minute when combined with stress. Stress can also interfere with body's self-repairing ability, including the innate potential of the heart to heal injuries. Depression and anxiety from stress tend to go together. Stress is an important risk factor for CVDs. All these suggest that an individual under constant stress can ultimately be susceptible to many ailments.

Emotional stress is associated with increased risk for cardiovascular disease. Chronic emotional stress has been linked to higher activity in an area of the brain which process emotions. It has been suggested that stress-reduction treatment along with pharmacotherapy significantly alleviate disease risk from emotional stress [1]. Women have been shown to be more vulnerable to stress and a study reported that sudden emotional stress can actually precipitate death in some women. Some may have a rare heart condition named Takotsubocardiomyopathy, or broken-heart syndrome, which is nine times more common in women than in men.

Psychosocial stress related to work is also a risk factor for chronic diseases. Job related strain and long working hours are associated with a moderately elevated risk for incident coronary heart disease and stroke. The relative risk differs based on sex, age, socioeconomic status etc. The benefit of reduction in work stressors in reducing the risk for CVDs is evident from studies. Many countries have launched healthy workplace campaigns, to prevent excessive work-related stress [2].

The society always considers children to be the most-risky population; they are hence usually provided extra protection. Children can also have stress. According to a report in International Journal of Adolescence and Youth, students in secondary and tertiary education systems face a wide range of chronic stressors related to academic demands [3]. Stress can reduce academic achievements, decrease motivation and increase the risk for drop out from school. Chronic stress in primary school students can play a major role in enhancing future physical, mental and social problems. It is vital that parents, families, teachers and professional health workers are

capable of detecting early symptoms of stress in children. An early intervention to reduce stress could help children cope with stress during transition to the adult stage and in adapting themselves to adult life and associated environs.

Given the increasing rate of silent heart attacks, strokes and other cardiovascular diseases in young adults it is important to find ways to reduce stress among people. Stress management tools are needed not only for people who have diseases linked to stress but also for healthy individuals for well-being.

Several stress management methods have been recommended. They include relaxation response method, bio-feedback, guided imagery (GI), diaphragmatic breathing, Transcendental Meditation,Cognitive Behavioural Therapy (CBT), Mindfulness-Based Stress Reduction (MBSR) and Emotional Freedom Technique (EFT). These strategies have been found to be effective foranxiety and depression in individuals. A wide range of biological effects such as reduction of cortisol levels in saliva, increased natural killer cells activity, decreases in blood pressureand heart rate, better cardiac rehabilitation, lesser psychological trauma and improved quality of life in patients after coronary artery bypass surgery, have been reported [4].

There also several other therapies suggested reducing stress. Laughter therapy, music therapy, pet Therapy and aroma therapyare some of the popular ones. Laughter therapist Keith Adams opines that laughter provides a full-scale workout for muscles and unleashes a rush of stress-busting endorphins. A patient who had a heart attack was found to have within a period of one year. fewer heart rhythm abnormalities (arrhythmias), lower blood pressure, lower blood levels of stress hormones and requirement for lower doses of medication, when he was advised to watch ‘humour’ videos for 30minutes every day.Aroma therapy using essential oil from basils, cassia, lavender etc., is also reported to produce effects such as decreasing systolic blood pressure and lowering blood levels of bad cholesterol.

Answer to the question posed in the beginning of this article is

uncertain. The human heart can manage infinite amount of stress depending on an individual's endurance levels. Practice healthy habits; live healthy, think healthy, work healthy and eat healthy. Our fist-sized heart if it is healthy can endure stress whatever its level may be.

It is not the burden that shatters you..
But the way you carry it...!!!
So better manage your Stress...
Before it begins to manage you...!!!
And Save your heart Because ;
Unfortunately you have only one...!!!!

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DOES THE BRAIN MATTER FOR THE HEART?

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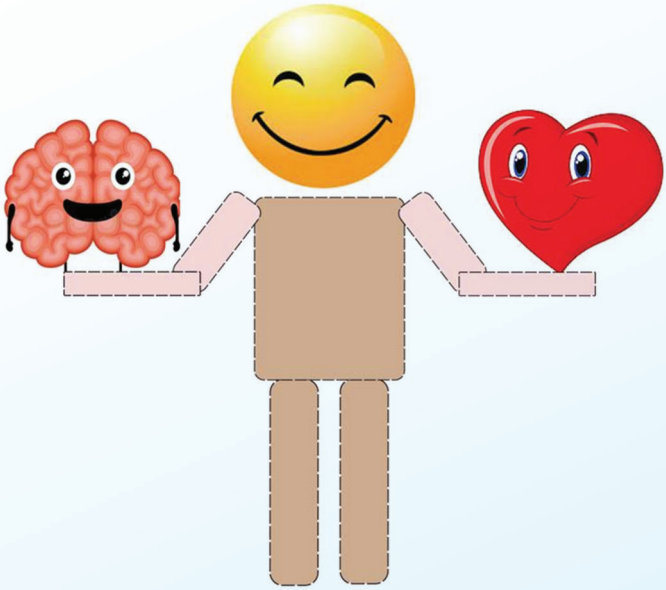
"silent, often subconscious conversation that is taking place inside us is one of the most vital communications we will ever find ourselves engaged in. It's the dialogue of emotion-based signals between our hearts and our brains, also known as the heart-brain connection" opines Professor Michael Miller at University of Maryland School of Medicine. We have known for decades that a complex interaction exists between the heart and the brain. They can intensively interact with each other to support bodily functions, but also can cause damage and lead to ill health. The heart provides proper perfusion of the brain whilea large network of cortical and subcortical regions of the brain control cardiovascular function. Hence, adysfunction in one system may lead to changes in the function of the other.

Earlier studies have delineated the basic structure of the autonomic nervous system (ANS), its divisions and its role in regulation of visceral and cardiovascular functions. Cannon in 1930s noted that



deaths from extreme emotions are likely because of hyperactivity of the sympathetic nervous system. Later electrocardiographic (ECG) studies, clinical observations and pathological assessments in patients with neurological disorders expanded our knowledge on neural regulation of cardiovascular function.From a clinical perspective, diseases affecting the heart–brain axis can be considered as either the effects of cardiovascular disease on the nervous system (such as cardioembolic strokes in patients with atrial fibrillation) or the effects of neurological disorders on the cardiovascular system (such as stress cardiomyopathy after aneurysmal subarachnoid hemorrhage [SAH]). The axis is regulated by the insular cortex in the complex network of cortical and subcortical regions of the brain. Neurological consequences of cardiovascular diseases also include cerebral ischemia and cognitive disorders.

Depression and cognitive impairment are the most frequent mental health problems among people with heart failure. Emotional stress has been identified as another key risk factor, accounting for about one-third of heart attacks and strokes. A heart attack is a result of sudden rupture of an unstable atherosclerotic plaque within a coronary artery. Plaque rupture can occur in stressful conditions. Release of biochemical compounds such as adrenaline

during severe stress raises heart rate and blood pressure and signals platelets to release a chemical, neuropeptide Y, which can cause spasm and transient occlusion of the coronary artery. Moreover, chronic mental stress can accelerate cardiovascular disease by promoting inflammation, oxidative stress, and abnormal function of the endothelium, the protective inner lining of our blood vessels.

Relaxation strategies can influence the heart–brain axis and thus improve biomarkers of cardiovascular risk. Patients with cardiovascular disease can use five strategies to reduce day-to-day stressors. These strategies include meditation (relaxation practices which activates serotonin), yoga (aids GABA induced mood stabilization), laughter (induces endorphin mediated visual effects), music (induces dopamine regulated auditory effects) and massages and hugging (activate oxytocin activated tactile responses). These techniques are expected to improve parasympathetic tone, the heart's ability to maintain blood pressure and/or heart rate during exposure to daily stressors.Listening to joyful music is considered to have cardioprotective and neurobiological effects, through reduction in systemic inflammation, blood pressure and heart rate, improved parasympathetic tone. Music therapy can be a useful intervention to speed recovery following stroke as dopamine is released in response to listening to or anticipating pleasurable music.

The time has possibly arrived to accept that effects of cardiovascular and neurological diseases may not be limited to the heart or brain.Given our expanding knowledge on the pathophysiology of the heart–brain axis and the interaction between the heart and the brain, there is a need for an integrative approach in treatment strategies for cardiovascular diseases and neurological disorders.

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NUTRITION MYTHS DEBUNKED!

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"If you have high blood pressure, reduce your salt intake"
"A low carb diet is the solution for your heart problems"

Every day we see headlines such as the ones shown above and which dictate what foods we should eat, why and how we should eat them. From social media to printed press, these fads dominate conversations, especially directed towards patients with heart disease, elevated cholesterol levels in blood, high blood pressure and obesity. Unfortunately, many of these diets restrict individual categories of food, instead of looking at food holistically. Below are a few nutrition myths debunked by scientific studies.

Avoid egg yolks if you have high cholesterol

While it is true that eggs contain cholesterol, the cholesterol that is associated with heart disease and plaque formation, is low density lipoprotein-cholesterol (LDL-C).Numerous studies have found that daily egg consumption does not necessarily lead to increased risk for coronary heart disease in middle-aged men and women. Eggs have

many other nutrients such as vitamins D, B12, B1, and B2, retinol, folate, ⍺-tocopherol, monounsaturated fat, saturated fat, linoleic acid, calcium and protein. The adverse effect of cholesterol in an egg is counterbalanced by potential beneficial effects of high-density lipoprotein (HDL) and triglycerides. Except in patients with type 2 diabetes, frequent egg consumption is not found to have any effect on blood levels of cholesterol [1, 2].

A No carb or low carb diet is good for reducing fat

Carbohydrates, more commonly known as "Carbs" seem to be a danger word in many diet prescriptions. Many diet prescriptionswhich promise accelerated weight loss within a span of few weeks often follow the no carbohydrates approach. The argument is that avoiding carbohydrates pushes the body into utilizing fats as an energy source.Carbohydrates are the body's preferred source of fuel, especially in the brain. Hence avoiding carbs can be an unpleasant and even dangerous approach to losing weight.

Low-carbohydrate diets may be accompanied by greater weight loss than other diets because of greater loss of water. Glycogen is more hydrated (2 to 3 grams of water) than fat (0.5 grams). The initial weight loss in people on a low carbohydrate diet is due to loss of body water induced by low insulin levels in blood. Minimal carbohydrate intake is also known to impair blood flow through coronary arteries, leading to damage of the heart muscles [3, 4].

Which fat is better: Saturated or Unsaturated?

It is known that fats are an important source of energy, but excess fat can actually build up in blood vessels and lead to chronic vascular and heart problems. High fat diets alone (keto diets) generally deplete muscle mass and facilitate rapid weight loss. Healthy fats are however important in a diet. Polyunsaturated omega-3 and omega-6 fats found in nuts, avocados and olive oil are in fact great for heart health [3, 4].

Whole food diets such as Whole30, Paleo etc.

These diets are the result of criticism against highly refined and ultra-processed food. They exclude refined items but also restrict

entire categories of food such as grains, cereals, dairy products etc. While these were advocated with good intentions, cutting off entire categories of certain food items is unhealthy and can cause an imbalance in nutritional levels. For some individuals, quitting cold turkey can result in psychological stress.

In conclusion, to have a healthy heart, eat lots of whole unprocessed food including but not limited to fruits, vegetables, nuts and grains. A balanced diet is the key. Avoid excessively oily, sugary and salty fried foods such as biscuits, chips, vadas and bhajis. It is okay to treat yourself once in a while. Focus on overall health rather than on losing weight and staying in shape by unhealthy means. Being heart healthy is not just about following a prescribed diet; it is a result of a combination of good food, regular exercise and frequent stress relief. Gratitude and happiness are also essential for good health.

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ANTIOXIDANT SUPPLEMENTS:
TOO MUCH OF A GOOD THING?

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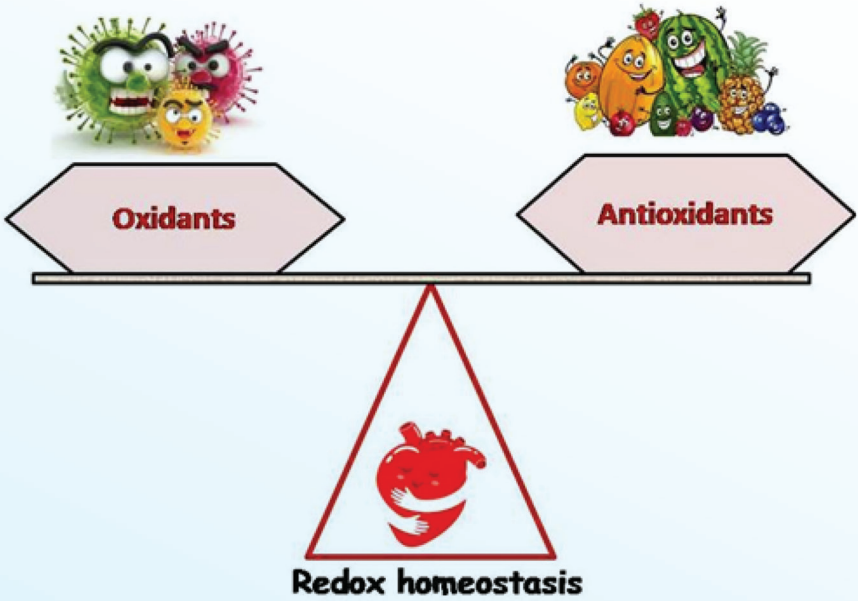
The word 'antioxidants' have become synonymous with good health. These supplements now line up the shelves of most medicals and supermarkets, promising to miraculously hold back ageing and chronic diseases especially heart diseases. It is known that several heart diseases are associated with high oxidative stress or simply a surplus of free radicals. When researchers first started linking free radicals with chronic disease, it seemed only logical that boosting our antioxidant intake, especially through supplements, would be the best antidote. Dietary supplements are the foundation of modern-day wellness regimens globally. Yet, as per recent research studies, in some instances excess supplementintake without medical supervisionmay do more harm to health than good.

The body responds to and tries to balance any increase in oxidative stress by producing endogenous antioxidants. Needless to say, antioxidants are really necessary to reduce cellular damage by free radicals in high oxidative stress conditions. This balancing strategy is called redox homeostasis. But what happens if the redox

'see-saw' gets too much altered, creating antioxidative stress? High levels of antioxidants could make atoms gain electrons, in a process called reductive stress. Dr Rajasekharan and his group at The University of Alabamaat Birmingham, have found that reductive stress is also pathological [1]. They report that a reductive stress can cause pathological heart enlargement and diastolic dysfunction (impaired filling of heart lower chambers). Their results from studies ina mouse model have been published in the June 2020 issue of Antioxidants and Redox Signaling.

Their findings indicate that chronic reductive stress is not tolerated and is adequate to induce heart failure. The study used transgenic mice that had upregulated genes for antioxidants in the heart, which increased the amounts of antioxidant proteins and reduced glutathione, the 'supreme antioxidant', thus mimicking a reductive stress condition. These mice had pathological heart changes and hypertrophic cardiomyopathy (resulting in abnormal thickening of walls of the ventricles - lower chambers - of the heart and thus reduced size of the cavity of the heart). They at six months of age had an abnormally high heart ejection fraction (percentage of blood leaving the heart on contraction) and diastolic dysfunction. Sixty percent of the high-reductive stress mice died before they attained eighteen months of age. When antioxidant glutathione production was temporarily blocked in a transgenic mouse with reduced free radicals, the heart changes could be prevented and the mice survived longer. This discovery may have clinical importance in the management of heart failure.

In another study, published in the journal Redox Biology, the impact of reductive stress on muscle stem (myo-satellite) cells was assessed [2]. These cells, located near skeletal muscle fibers are able to regenerate and differentiate into skeletal muscle after chronic and acute muscle injury. Investigators found that tilting the redox see-saw to oxidative stress, reduced the regeneration of skeletal muscle; altering the redox to reduced stress also caused significant inhibition of muscle satellite cell differentiation. This study did not however look into the effects of externally ingested antioxidants from fruit and vegetables on health outcomes.



There is a general misconception that free radicals are bad, and that antioxidants are super foods. A 2019 CRN Consumer Survey on Dietary Supplements found about 77% of Americans consume dietary supplements every day. Within this group, 58% consume antioxidants as multivitamins [3]. Of course, like most factors that affect our health, the case for antioxidants is not simple. In this scenario, caution must be exercised, as long-term consumption of antioxidant drugs by any individual without assessment of their redox state could result in reduced stress, which can alter normal body mechanisms and slowly damage heart and skeletal muscles. The indication from studies cited that "eat a proper diet that contains a variety of antioxidants, but do not go overboard".

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A BIOELECTRONIC DEVICE TO MONITOR HEART ATTACK

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Researchers at Tufts University have developed a small device with networks of channels of microliter dimensions and a tiny computer chip, that monitors the real-time electrical and physiological changes that occur in the heart prior, during, and after a heart attack.

A heart attack is a consequence of blockage in arteries that supply blood to heart muscles. Arterial block and loss of blood supply result in oxygen deficit (hypoxia) and lead to tissue death, abnormal electrical and physiological changes, and decline in the function of the heart. because of Biological and electrical activity are critical determinants of healthy heart function. Organized electrical impulses maintain the synchronized pumping or contraction-relaxation cycle in the heart. Oxygen levels drive the metabolic activity and functions of ion channels in the heart. Electrical measurements of the shape, size, and duration of action potential (voltage change across cell membrane) provide indirectly, information on the metabolic and functional state of the heart.

Hypoxia causes rapid changes in the electrical and physical activity of the heart cells leading to deterioration in the heart function. These changes in the heart under hypoxia on a real-time basis cannot be presently studied.

A group of researchers in the Department of Biomedical Engineering at Tufts University School of Engineering, Massachusetts in the United States. has reported in the journal Nano Letters evidence that the electrical and physiological responses of the heart under hypoxic conditions can be monitored using a biosensor chip during and after a heart attack.

The fully assembled chip is integrated with bioelectrical devices, which can be placed both inside and outside the heart muscle cells. Platinum-based electrodes placed inside the heart cells enable continuous recording of voltage change across cell membrane before, during, and post hypoxia induced by arterial block. Recording elements that allow the chip to record the beating rate, rhythmicity, and propagation of an electrical impulse in the heart cells are assembled outside the cells.

In the chip that has been developed, heart cells supplied with a fluid for nourishment of cells, rest on platinum-coated nanopillars/platinum-tipped needles. The flow of fluid which bathes the cells can be controlled and thus create normal oxygen (21%), reduced oxygen (1-4%), and recovered oxygen (21%) conditions which are similar to what we observe in normal heart, during a heart attack, and after a heart attack. When an electric current is supplied across the cells, tiny needles that penetrate the cell membranes record time-lapsed and multiple readouts. A two-dimensional predictable wave pattern was observed when voltage

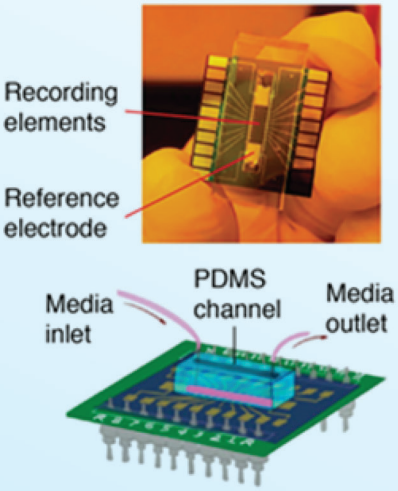

Figure. Assembled chip with integrated recording elements.

waves passed over the heart cells. There was however slower and erratic wave pattern when heart cells were supplied with reduced oxygen levels. The electrophysiological properties of heart cells thus could be monitored in a continuous manner using the newly developed bioelectronic microfluidic chip.

The inventors have extended the use of their device to enhance new treatment strategies and discovery of novel drugs for patients suffering from heart attack. In the future, other than oxygen deficit, factors such as higher acid levels, waste accumulation and higher potassium levels that contribute to heart attacks could also be studied by altering various features of the chip.

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IS SOCIAL DISTANCING A NEW CHALLENGE FOR HEART HEALTH?

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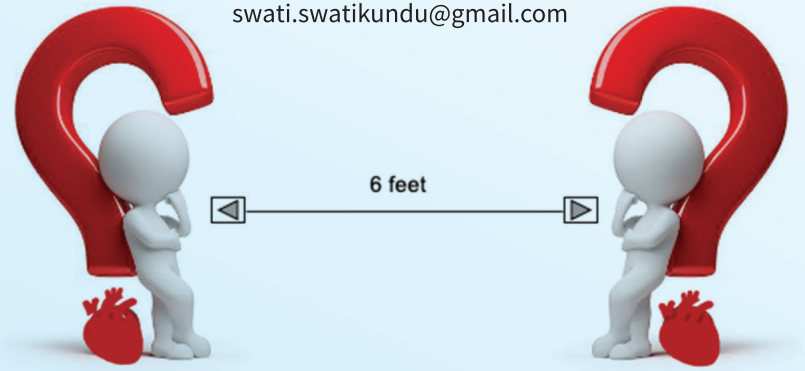

As a cardiovascular scientist and actively evaluating the effects of COVID-19, it is inevitable for me to draw a connection between viral infection and heart health. As the viral pandemic evolves, we are learning more about its indirect effects and target audience. From the beginning itself, it was known that old age groups and people with existing medical conditions including heart diseases are at a higher risk. A study suggests that

more than 40% of critically affected patients with COVID-19 had pre-existing cardiovascular conditions [1]. This may be because the viral cells attack endothelium (inner layer) of blood vessels and, therefore, results in plaque formation which leads to cardiovascular disorders (CVDs) [2, 3].

There is no denial that COVID infection poses a higher risk to people with existing CVDs. These people are hence advised to practice greater caution. These measures include now-a-norm social distancing. While focusing on the prevention of viral infection, what has missed the eye is the detrimental effect of social distancing measures on heart health. As the social distancing guidelines are here to stay for long, we must address its significant and lasting adverse impact on our heart health.

The mechanism underlying the effects of social distancing on human heart remains undiscovered; however, a study reports an association of social distancing with 29% of incidence of coronary heart diseases and 32% of incidence stroke [4]. Persistent social stress resulting from social distancing activates a central stress response--the hypothalamic-pituitary-adrenal axis [4, 5]. The other system which gets affected is the sympathetic nervous system. Its activation leads to multiple cardiovascular effects including heart rhythm disorder such as spontaneous ventricular arrhythmias, ventricular tachycardia, and myocardial electrical instability [6]. In addition to this, stress from social distancing also exerts adverse effects on autonomous homeostasis which in turn results in inflammation and endothelium dysfunction [7]. Eventually, endothelium dysfunction leads to a vasoconstrictive state with increased cell adhesion and oxidative stress resulting in the development of atherosclerosis.

Since CVDs are the leading cause of death globally [8], we should not lose sight of heart health while taking all the measures to prevent COVID. As we are not sure how long we need to follow the distancing guidelines, it is essential to get physically active to mitigate psychological effects of social distancing. It is time to admit that socialising has a pivotal role in keeping hearts healthy in some way.

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HYDROXYCHLOROQUINE IS ASSOCIATED WITH INCREASED RISK FOR CARDIAC ARRHYTHMIAS

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In a novel study at Beth Israel Deaconess Medical Center (BIDMC) a group of clinicians have observed that patients who were administered hydroxychloroquine for COVID-19, were at increased risk of electrical changes to the heart and cardiac rhythm disorders (arrhythmias) [1]. When hydroxychloroquine was administered along with azithromycin to COVID 19 patients with heart disease, there was longer time to recharge between two heart beats in them, when compared to patients with COVID 19, who were not administered the drugs.The changes could be seen on by analysis of the electrocardiogram (ECG).

World Health Organization (WHO)has announced COVID-19 as a pandemic disease. More than 43 lakhs of people worldwide have tested positive for the infection and more than 5 lakhs have died till date. There is no FDA approved treatment available for COVID 19.

Hydroxychloroquine, one of the most important antimalarial drugs is used as a potential therapy for the pneumonia that accompanies COVID-19, with or without the antibiotic azithromycin, irrespective of patients having other chronic diseases. A study in a small number of patients with COVID-19 observed benefit from this anti-malarial drug.

At BIMDC, clinicians studied a cohort of patients hospitalized with COVID 19 at an academic tertiary care center in Boston, Massachusetts. The cohort included patients with heart disease, hypertension and diabetes mellitus. Patients were administered 400 mg of hydroxychloroquine twice on day 1 and then 400 mg daily on days 2 through day 5 along with azithromycin.An increased frequency of adverse drug events was observed when compared to patients with pneumonia only. On hydroxychloroquine and azithromycin administration, there was an electrical disturbance in the heart indicated by a longer space between specific peaks, observable on an electrocardiogram. This finding denotes that the heart muscle is taking milliseconds longer than normal to recharge between beats. The delay can cause cardiac arrhythmias, which in turn can increase the probability of a cardiac arrest, stroke or death. Malaria Policy Advisory Committee Meeting at WHO has suggested that hydroxychloroquine is structurally and functionally similar to the class IA antiarrhythmic quinidine, which inhibits voltage-gated sodium and potassium channels, and can lead to prolonged QT interval and increase the risk for sudden cardiac death [2]. Pharmacy specialist, Nicholas J. Mercuro at BIDMC opines that “This is especially concerning, given that patients with underlying cardiac co-morbidities appear to be disproportionately affected by COVID-19 and that the virus itself may damage the heart”[3]. The study at BIDMC study indicates that caution should be exercised before administering hydroxychloroquine to patients with COVID-19. “If considering the use of hydroxychloroquine, particularly combined with azithromycin, clinicians should carefully weigh the risks and benefits, and closely monitor medication interactions, cardiac manifestations, routine electrocardiograms, and electrolyte monitoring particularly considering patients’ co-morbidities and

concomitant medication use,"states Howard S. Gold, an infectious disease specialist at BIDMC.

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