# Myocarditis: Once Rare, Now Common

01/05/2023 by Dr. Thomas Levy

OMNS (Jan. 5, 2023) As an actively practicing clinical cardiologist for many years in three different communities, I knew about myocarditis. I just never saw it. Quite literally, I recall seeing ONE young woman who presented with a picture of acute congestive heart failure, and her echocardiogram study revealed a big and poorly contracting heart. Such a condition is diagnosed as an idiopathic congestive cardiomyopathy, which basically means the heart is enlarged and functioning very poorly, and you have no idea why. After treating her with traditional measures for congestive heart failure, she started getting better. To my great surprise, after six to nine months of follow-up, her echocardiogram had returned to normal.

Retrospectively, it was then clear that she had likely contracted a virus that focused on her heart. The virus-induced inflammation in her heart muscle cells then decreased the strength of her heart contractions to the point of clinical heart failure with heart enlargement. Presumably, her young immune system eventually "kicked in" and eliminated the viral culprit. Even as a clinician who also received many patients in consultation from other doctors, she represented the ***entirety*** of my cases of myocarditis. And at that, the diagnosis was only a retrospective conclusion.

## COVID and Myocarditis

Today, the active clinical cardiologist is seeing myocarditis patients on a regular basis. The scientific literature indicates that myocarditis is occurring quite frequently in patients harboring the chronic presence of the COVID-related spike protein. This is being seen in many individuals with persistent chronic COVID, many of whom have been vaccinated, as well as in a substantial number of individuals who have been vaccinated and have never contracted COVID. [1-4] A study in mice showed that the injection of the mRNA vaccine (which produces the spike protein) reliably induced myopericarditis. [5] Regardless of the initial source of exposure to spike protein, it appears to be the reason for the pathology and symptoms seen in chronic COVID. [6]

While not yet clearly documented by any well-designed studies in the medical literature, a great deal of anecdotal information indicates that vaccine mRNA shedding can occur. And once transmitted, the mRNA directly leads to spike protein production. [7] Such mRNA shedding means that the spike protein is indirectly, if not directly as well, transmissible from one individual to another via inhalation or various forms of skin contact. In fact, Pfizer's own internal documents advise about the possibility of "environmental exposure" by "inhalation or skin contact" of the mRNA in the vaccine being transmitted from a vaccinated individual to another person. [8] Furthermore, while many try to dismiss such an "exposure" as too minimal to be of clinical consequence, such an assertion cannot be assumed to be true when dealing with an agent (spike protein) that appears capable of replication once it gains access to the body. The toxicity associated with spike protein would not be due to a one-time exposure, but one that could persist indefinitely because of this ability to replicate. A toxin that has such an ability is truly a clinical nightmare. It is never a good idea to overestimate the integrity of the pharmaceutical industry. [9]

The spike protein is the part of the COVID pathogen that facilitates its entry into various cells in the body. [10] This cellular entry occurs after the spike protein binds to ACE2 receptors present on the cell membranes found in a wide variety of tissues and organs. Spike protein binding to ACE2 receptors in the lungs, heart, and blood vessels has proven to be of particular importance in determining the severity of many COVID infections as well as the nature of the side effects seen following a spike protein vaccination. Deaths and severe complications have also resulted from vaccine-induced thrombosis occurring in the cerebrovascular circulation. [11,12] Autopsy evaluation of multiple vaccinated individuals who died shortly after receiving their vaccinations revealed acute myocarditis as the only logical cause of their deaths. [13]

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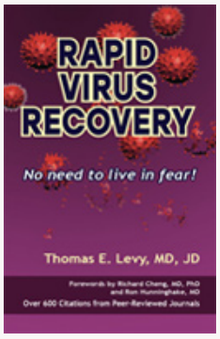


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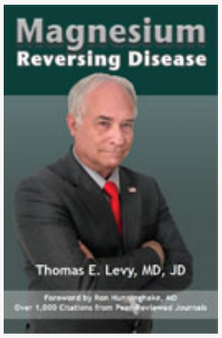


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Sufficient spike protein binding to ACE2 receptors on the endothelial cells lining the blood vessels has consistently resulted in increased blood clotting. Such clots are tiny in some people, which can then lead to various degrees of tissue and organ damage depending on how severely overall blood flow is impaired to those areas. [14,15] Other clots can rapidly increase in size and result in sudden death. [16] Spike protein can activate blood clotting by binding directly to the ACE2 receptors of platelets in the blood. [17,18] Also, circulating spike protein that has not yet been bound appears to stimulate hypercoagulation as well. [19] Of note, both Pfizer and Moderna appear quite proud to assert that their final formulations supply the "full-length" spike protein in the injections.

Myocarditis, which simply means inflammation of some or all of the muscle cells in the heart, can occur when the spike protein binds to the blood vessels in the heart, to the muscle cells themselves, or both. [20] Even when the myocardial blood vessels get more selectively targeted, inflammation of the heart muscle itself will still eventually ensue as the circulation of the heart gets progressively impaired by blood clotting and/or by an increased resistance to blood flow resulting from inflammation-induced vasoconstriction. Pre-pandemic myocarditis (cases not related to a spike protein presence) generally did not involve any predisposition to blood clotting in addition to the inflammation of the affected heart muscle cells.

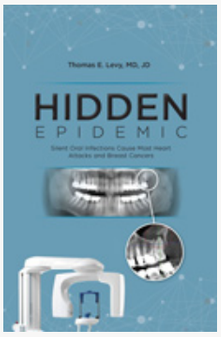
Myocarditis presents no diagnostic challenge when it presents in its classical manner. Chest pain and rapid heart rate are often the earliest symptoms. If the myocardial inflammation is evolving rapidly, symptoms of congestive heart failure, including shortness of breath and swelling of the lower legs, can occur as well. Not uncommonly, an upper respiratory tract viral infection will be present or there will be a history of such an infection having recently resolved. Chest X-ray, electrocardiogram (ECG), and echocardiogram can all be used to help establish the diagnosis. An elevated troponin level on blood testing is extremely sensitive in picking up any ongoing heart muscle cell damage, and some elevation of this test will always be seen if any significant inflammation is present in those muscle cells.

> Any continued elevation of troponin in the blood, ***however minimal***, must be regarded with significant concern, even if there appears to be a complete clinical resolution of the myocarditis. Everyone should have this test done, even if they are feeling perfectly well, to both establish a baseline within the normal range or to detect any unsuspected low-grade myocardial inflammation.

The very high sensitivity of the troponin test has revealed that there are countless numbers of people post-COVID infection and/or post-vaccination that are continuing to have sustained subclinical degrees of myocardial inflammation. No matter how minimal the elevation of the test, any increase means that a gradual and continued loss of heart muscle function will occur over time. It also means that the heart is highly susceptible to an acute and potentially severe worsening of heart function when an additional exposure to more spike protein occurs, as is seen with the booster shots being vigorously promoted now. A heart with a minimal elevation of troponin is literally the perfect setting for a catastrophic clinical response when an additional spike protein-laden injection is given, much like what gasoline would do to smoldering coals. Not surprisingly, it has been shown that COVID patients with higher troponin levels were more likely to die than those with lower levels. [21]

Many abnormal troponin tests eventually resolve completely and ***many do not***. The quality of nutrition, the strength of the immune system, and the quality of the nutrient/vitamin/mineral supplementation being taken are all critical factors in determining whether a minimal, subclinical degree of inflammation in the heart is capable of completely resolving with a return of the troponin level into the reference, or normal, range. With much of the world eating poorly and not supplementing at all, there is an ongoing presence of the spike protein in a ***very large*** number of people around the world. Clinical myocarditis is simply an ***advanced*** state of inflammation in the heart, with much higher levels of troponin being released into the blood. Cardiac injury was detected in 20% to 40% of patients hospitalized with COVID. [22,23] Any troponin elevation in hospitalized COVID patients was associated with an increased mortality. [24]

Troponin testing is currently the most important and widely accepted way to determine whether a suspected heart attack has occurred, with the troponin being released into the circulation as the heart muscle cells die. [25] Some degree of myocardial injury is felt to be present when any troponin level is detected beyond the 99th percentile upper reference limit, whether in the context of a suspected heart attack or the possible presence of any inflammation in the heart. [26,27] Even an increase in baseline troponin levels that remains below the established upper limits of normal has been shown to be significantly associated with increased mortality after noncardiac surgery. [28] Baseline troponin testing is a good idea for everyone, since normal ranges can vary from lab to lab, and because it appears myocardial injury can still be present when the troponin level rises significantly from a baseline point but

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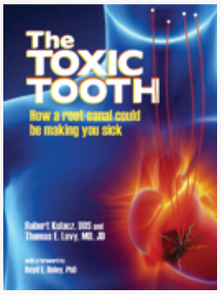

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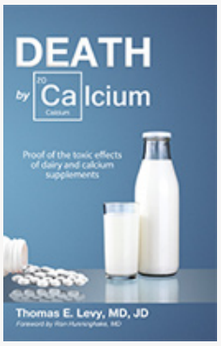

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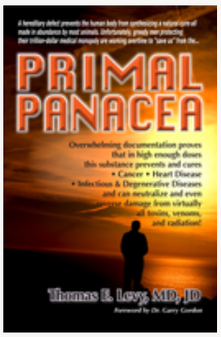

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remains short of the upper reference limit. [29]

The importance of the most minimal of troponin elevations has been established in several studies looking at the relationship of pre-operative troponin levels with long-term mortality following noncardiac surgery. Compared to patients with no troponin elevation, a significant increase in 30-day mortality was seen in patients having minor troponin elevations following noncardiac surgery. [30,31] Another similar study found over a doubling of the mortality rate when the two patient groups were evaluated at three years following the noncardiac surgery. [32]

In a recent Swiss study yet to be published at the time of this writing, troponin levels were measured on 777 hospital employees who received a booster injection after having received two shots previously. On the third day after the booster, troponin levels above the upper limits of normal were seen in 2.8% of those subjects. By the next day, half of the elevated troponin levels had come back into the normal range. [33] Longer-term follow-up data was not available. This study raises more questions than it answers. What would the troponin levels have been at one day post-injection? Did the troponin levels still elevated at day four post-injection resolve completely? If so, how long did that take to occur? Rather than be concerned that some myocardial damage was done by the vaccine, which is openly acknowledged in the study, it is dismissed as being of no importance since half of the elevated troponins resolved 24 hours later. And, as with all of the current papers downplaying the significance of any vaccine side effect, however significant, the authors always conclude that the vaccine is doing much more good than harm without any further qualification as to why such a conclusion is valid.

Having even the most minimal elevation of troponin not only raises the concern of some collective long-term heart damage, or the ease of having a "re-flaring" of inflammation with new spike protein exposures, as from a booster shot, it also raises the concern of electrical instability in some of the inflamed myocardial cells. There is always a possibility of electrical instability in any inflamed myocardial muscle cells, as it is their normal physiological nature to transmit electrical impulses from one cell to the next. Because of this, stressful events that release surges of adrenalin and catecholamines in the circulation, as is seen with peak physical exertion, can readily provoke such electrically unstable cells into starting, and sustaining, an abnormal heart rhythm. Literally hundreds of European soccer players have died or collapsed on the field of play in the last two years. Of note, they have not been seen to collapse while standing or sitting on the sidelines. Similarly, any pilot with even a minimal but otherwise symptom-free elevation of troponin can potentially sustain such a life-threatening arrhythmia when a significant stress-provoking emergency arises in the cockpit.

However, regardless of any benefits a COVID vaccine might have on the overall morbidity and mortality on those receiving it, it completely ignores that MANY effective treatments have emerged that either prevent most cases of COVID or readily cure them when properly applied after the infection has been contracted. [34-38]

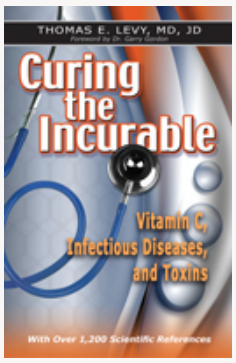
> With the availability of effective treatments, ***no*** vaccine side effect, especially one that has already resulted in many deaths, should be tolerated, unless the vaccine candidate is fully aware of all possible side effects and chooses not be bothered with measures proven to prevent and/or treat the infection.

To date, every vaccine that has ever existed has a significant side effect profile. This information, along with a full disclosure of effective non-pharmaceutical therapies for the condition the vaccine is supposed to prevent, should ***always*** be afforded to both physicians and their patients.

It is important to realize that most of the tissues and organs of the body do not have reliable laboratory markers indicating the presence and degree of ongoing spike protein damage. Tracking heart damage with troponin levels makes this organ relatively unique in this regard, and since ACE2 receptors are present in most organs and tissues, any continued elevation of troponin can also be considered a reliable indicator that spike protein damage is occurring in organs and tissues outside of the heart. Spike protein would be expected to bind ACE2 receptors wherever it finds them, and such binding would always be expected to cause cellular inflammation and damage. Blood testing for natriuretic peptides also reflects myocardial damage, but the primary focus should remain on troponin testing and doing whatever is necessary to return that test into the normal range. [39-45]

## COVID, Arrhythmias, Heart Block, and Pilots

As would be logically expected, any agent that can cause inflammation in the heart would also be expected to sometimes involve the cells in the heart that generate and conduct each electrical spark that initiates every contraction of the heart. As myocarditis can be patchy and not affect all of the heart muscle cells uniformly, heart rhythm problems are not always part of the clinical presentation of myocarditis. However, various degrees of heart block have been reported because of the COVID-19 infection and/or because of the COVID-19 vaccination. [46-51]

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A new condition known as multisystem inflammatory syndrome in children (MIS-C) has emerged since the onset of the COVID pandemic, appearing primarily in advanced COVID infections. [52,53] MIS-C, and MIS in adults, simply means the COVID infection has resulted in a widespread amount of inflammation in the body, often involving the heart and the lungs. Minimal to advanced heartbeat conduction problems have occurred secondary to MIS-C, ranging from the often-innocuous prolonged PR interval (see below) on the ECG to advanced and potentially life-threatening degrees of AV block. [54,55] When heart function is normal, the AV node allows a rapid conduction of the heartbeat throughout all of the heart muscle cells so that heart muscle contraction is synchronized and optimally efficient. AV block results in an abnormal slowing of the heart rate and sometimes fatal secondary arrhythmias, including complete stoppage of the heartbeat (asystole). It appears likely that the spike protein can damage the heart at any age, and that the spike protein can be present because of the infection itself and/or the vaccination targeted at the infection.

The PR interval is the amount of time that the heartbeat takes to traverse the atrial chambers in the heart before reaching the conduction-accelerating AV node. The normal PR interval ranges from 0.12 to 0.2 seconds. In younger individuals, especially well-trained athletes, a PR interval greater than 0.2 is usually completely normal. However, when PR interval measurements have always been 0.2 or less and ***then*** start to lengthen as an older adult, there should be significant concern that the aging conduction system might manifest more significant conduction abnormalities in the future.

In the setting of the pandemic, it is of particular concern when PR interval prolongation is seen for the first time following a bout of COVID and/or following a vaccination. This is a ***clear indicator*** of new inflammation in at least some of the heart cells, however minimal it may be. Regardless, it should not just be assumed to be of no importance. All disease has a spectrum of pathology, and the earliest stages of pathology should never be trivialized. [56] In a Harvard study that extended over a 30- to 40-year period, it was found that individuals with PR intervals greater than 0.2 seconds had ***twice*** the risk of atrial fibrillation, ***three*** times the risk of needing a pacemaker (meaning the presence of advanced degrees of heart block), and nearly a ***one and a half*** times increase in all-cause mortality. Furthermore, greater degrees of PR interval prolongation led to an even greater risk. [57]

However, ignoring the inherent pathology in a pandemic-induced prolonged PR interval is exactly what the Federal Aviation Administration (FAA) appears to have done. Facing a shortage of pilots due to both the vaccine requirement it initiated during the pandemic for the pilots to fly, along with many early retirements that resulted, the FAA decided to change the rules, disregarding long-standing parameters of normalcy based on medical science and not convenience. The FAA has now ***declared*** a PR interval of 0.3 seconds to be the "new normal" in the FAA Guide for Aviation Medical Examiners as of October, 2022. The October, 2021 standards asserted the PR interval was normal only at 0.2 seconds or less. When the pilot has "no symptoms" he or she can now obtain clearance to fly with a PR interval of 0.3 or less. And when that interval is greater than 0.3, a "current Holter and cardiac evaluation" are then required. Considering that the normal PR interval ranges between 0.12 and 0.20 seconds, an interval of 0.3 seconds represents a "permissible" increase in this interval by ***over 100%*** relative to the low normal interval of 0.12 seconds. This is not a nominal increase in PR interval, but a very large one.

Even now, a treadmill exercise stress test is not required to receive medical clearance to fly, even for commercial pilots. This is simply not a safe policy by the FAA and arguably a ***shocking*** one, as many pilots are in the age range when heart attacks occur without any early symptoms but with a normal ECG, the ECG being the only mandatory heart-related test. Roughly a third of all deaths around the world are due to cardiovascular disease. And in western countries sudden cardiac death occurs in about half of all coronary artery disease patients. [58,59] Much more vigorous cardiac evaluations should be performed in prospective pilots, and repeated at appropriate intervals. A normal ECG means a heart attack has not occurred, ***nothing more***. A fatal heart attack from very advanced coronary artery disease could occur 10 minutes after the normal ECG was recorded. No pilot should ever fly when there is a persistent elevation of troponin levels ***and/or*** D-dimer levels (see below). It is irrelevant that the pilot might feel well, have a normal ECG, and have no clinical evidence of myocarditis.

## COVID, Blood Clots, and D-dimer Levels

A D-dimer blood test is a measure of the degree to which blood clots already formed are breaking up (lysis) and releasing those breakdown products into the blood. It is not a measure of how prone the blood is to clotting in the first place (increased coagulability). However, it is a very sensitive test that will always be elevated when increased blood clotting is taking place, since those clots must still be broken down to keep the circulation from shutting down. Except when elevated in the setting of a very minimal number of chronic diseases, an elevated D-dimer test very reliably means there are blood clots breaking up because too many new blood clots are continuing to be formed. Only rarely is significant thrombosis seen in the absence of an elevated D-dimer level. [60]

In the setting of the pandemic with a history of active or chronic COVID infection, as well as a history of having had one or more vaccinations, an elevated D-dimer test is always a cause for GREAT concern. It is clear-cut evidence that there is an ongoing spike protein presence binding ACE2 receptors in the inner lining (endothelium) of blood vessels in the body, resulting in platelet activation and subsequent blood clotting. [61] Blood clots can range from microscopic to massive. Such clotting can also be part of a myocarditis presentation, although not necessarily so. Certainly, having both an elevated troponin level and an elevated D-dimer level is especially worrisome and warrants prompt treatment in order to normalize the pathology causing them.

Both the COVID vaccine and the COVID infection have been documented to cause increased blood clotting and thrombosis. [62,63] Viral infections in general have also been found to cause abnormal blood clotting. [64] In critically ill hospitalized COVID patients, elevated D-dimer levels were found about 60% of the time. [65] Not surprisingly, the longer that D-dimer levels remain elevated in COVID patients, the greater the morbidity and mortality. [66-68] Similarly, the higher the D-dimer level on hospital admission for COVID, the greater the chances of in-hospital mortality. [69]

When the underlying infection or other pathology can be resolved, D-dimer levels will generally resolve as well. If a thrombotic event occurs, resolves, and has no ongoing underlying pathology, D-dimer elevations will generally persist for only a few days before returning to normal. Chronic COVID infections often demonstrate persistent blood clotting problems. In one study, 25% of a recuperating group of COVID patients who were four months past the acute clinical phase of their infections demonstrated increased D-dimer levels. Also of note, the other common laboratory parameters of blood clotting had already returned to normal in over 90% of the patients, indicating the sensitivity that D-dimer testing has for detecting the blood clotting pathology. These other tests included prothrombin time, partial thromboplastin time, fibrinogen, and platelets. Even C-reactive protein and interleukin-6, tests that track inflammation, had typically also returned to normal. [70]

Platelet levels generally drop in the blood at the same time D-dimer levels are increasing, as they are consumed in the formation of the blood clots. [71] A post-COVID vaccination syndrome known as vaccine-induced prothrombotic immune thrombocytopenia (VIPIT) with these laboratory findings has been described. [72-75]

While the pandemic has given more attention to D-dimer testing than it ever had before, other conditions can cause a D-dimer elevation. [76] However, anyone today who is not acutely ill but found to have an elevation of their D-dimer levels is likely suffering from the consequences of persistent spike protein presence in their vasculature, whether due to lingering COVID infection and/or due to having received one or more COVID vaccinations. And even if such an individual never had COVID or received a vaccination, an extensive medical evaluation is warranted, since a D-dimer elevation is never normal. A persistently elevated D-dimer levels should never be dismissed as inconsequential just because the patient feels well.

## Therapeutic Recommendations

Quite simply, the goal is to normalize both troponin and D-dimer levels in everyone under treatment. This can be more difficult to achieve in older patients with chronic medical conditions that are being clinically managed. But a concerted effort should still be made at the outset to normalize these tests.

Nearly all of the elevated troponin and D-dimer levels at this point in the pandemic will be secondary to persistent spike protein presence in the body following COVID infection, one or more COVID vaccinations, or both. The likely ease of spike protein transmission also means there will be some individuals who have elevated test levels without having knowledge of ever having been infected, and without a history of vaccination. In other words, these tests ***should be performed in everyone*** at this point in time, and any elevations should be aggressively treated. And if those tests are completely normal, they will still serve as excellent baseline data when dealing with future medical conditions or infections, COVID-related or otherwise.

> There is no one set protocol for dealing with a persistent spike protein syndrome with elevated troponin and/or D-dimer levels. Some individuals will respond quickly and regain a normal health status after relatively minimal measures are taken. Others will require very aggressive and prolonged treatments, and still others will simply not normalize regardless of what is done. In younger patients, the inability to regain a normal health status should be extremely rare, especially when a quality regimen of nutrients, vitamins, and minerals is being introduced for the first time.

The following recommendations apply to an individual with elevated troponin and D-dimer levels, or with either one elevated and the other normal. Specific reference ranges, or normal ranges, for these tests should come from the laboratory running the tests, since significant variation in these ranges can be seen from one testing source versus another. These recommendations apply to both the clinically normal individual and someone who is suffering from chronic COVID or any of a variety of nonspecific symptoms. This protocol, and all variations thereof, should be administered with the guidance of a licensed healthcare professional.

1. Intravenous vitamin C, dosed roughly between 50 and 150 grams (1 gram/kilogram body weight),

infused over 60 to 120 minutes. Add 25 mg of hydrocortisone to each IV. If not available, take 50 mg of hydrocortisone orally about one hour before start of infusion. Also add 500 to 1,500 mg of magnesium chloride to each IV bag. For more information on vitamin C administration: [77]

Alternatively, take 5 packets of LivOn Labs liposome-encapsulated vitamin C orally three times daily. [78] If available take 10 to 20 mg of hydrocortisone orally with each dose.

Alternatively, 2 to 4 grams of sodium ascorbate in juice three times daily with 10 to 20 mg of hydrocortisone with each dose.

2. Follow each vitamin C infusion with a separate infusion of methylene blue [a potent anti-pathogen proven to be of great benefit even in the most advanced stages of COVID] [79-84]:

   50 mg of MB in 250 ml of 5% dextrose solution can be infused over 30 to 45 minutes.

   Alternatively, 50 mg of MB can be taken orally each day of vitamin C administration. 5 ml of 1% MB solution in juice (tomato a good option). Taking through a straw avoids temporary teeth and tongue staining. Prompt administration of 3% hydrogen peroxide removes skin stains.

3. Hydrogen peroxide nebulizations as tolerated to eliminate low-grade colonizations of COVID and other pathogens in the aerodigestive and lower digestive tracts. [85]

Any, or all, of the following nutrient/vitamin/mineral supplements for general support of long-term health: [86]

- Vitamin C
- Magnesium chloride
- Zinc and quercetin
- Vitamin D
- Vitamin K2
- Olive leaf extract
- Multivitamin, multimineral preparation that has no added calcium, iron, or copper
- Nattokinase, lumbrokinase, and/or serrapeptase to minimize any future blood clotting problems

At the discretion of the healthcare professional, any of the following measures can be added:

- Ozonated blood or ozonated saline infusions
- Ultraviolet irradiation treatments of the blood
- Intravenous infusions of hydrogen peroxide
- Hyperbaric oxygen treatments
- Chlorine dioxide treatments
- Hydroxychloroquine or chloroquine
- Ivermectin

Any modifications of these treatments, along with deciding how long they should be continued, must be determined on an individual basis with the help of the chosen healthcare professional working with the patient.

## Recap

Myocarditis was once rare. Because of COVID vaccines and COVID itself, myocarditis has become genuinely common. The troponin test has shown that there are many individuals who continue to have low-grade myocardial inflammation after a return to clinical normalcy. This makes such individuals ticking time-bombs ready to develop a serious worsening of their underlying pathology when a booster shot is received or a recontraction of COVID or one of its variants occurs. The persistent inflammation in the heart means there is a persistence of the spike protein in that organ and very likely through much of the body. This sets the stage for a sudden and dramatic decline in health when more spike protein is administered or allowed to be replicated in the body.

Elevated D-dimer levels indicate an overactive state of blood clotting in the body, and when these levels remain elevated, the long-time prognosis is likely very poor in terms of morbidity and early mortality.

Heart rhythm problems and heart block can occur when troponin levels remain elevated. The FAA is currently changing its rules to allow more pilots to fly who have PR interval greater than 0.3 seconds, a development that should be of great concern to all who fly. PR intervals that lengthen in the older population can presage significant heart problems, including early death. Science should never be displaced by political expedience and the need to make ever greater amounts of money.

Any persistent elevations of troponin and D-dimer tests must be treated with the goal of normalizing them completely. Obviously, this is especially important in the pilot population. Measures to accomplish this along with recommended types of long-term supplementation are discussed.


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