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The Mammalian Stress Mechanism Explains Covid, Long Covid and Sudden Death

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Abstract

Mammalian stress mechanism (MSM) hyperactivity explains the bewildering local and systemic manifestations of coronavirus infestations. The "normal" coronavirus attacks the pulmonary endothelium and causes mild and transient localized MSM hyperactivity that exaggerates exudates which cause coughing and sneezing that spreads the contagion. It also exaggerates the "leakage" of pulmonary tissue factor into systemic circulation, causing mild systemic MSM hyperactivity that explains the fever, fatigue, and malaise of the common cold. The exaggerated virulence of the "novel" coronavirus causes severe pulmonary MSM hyperactivity, which generates excessive exudates that disrupt gas exchange in the lung and manifests as ARDS (acute respiratory distress syndrome). It also causes extreme leakage of pulmonary tissue factor into systemic circulation, causing severe systemic MSM hyperactivity that manifests as loss of smell, taste and hair as well as fever, fatigue, and malaise. Both normal and novel coronaviruses remain confined to the lung, but the mRNA "immunization" introduces the coronavirus "spike protein" directly into systemic circulation, where it proliferates and attacks the vascular endothelium throughout the body. The resulting systemic MSM hyperactivity secondarily elevates nonspecific immune activity but fails to elicit specific immune activity that conveys lasting protection. Meanwhile, the systemic disruption of the vascular endothelium causes pericarditis, myocarditis, and infertility, as well as fever, fatigue, malaise, and loss of smell, taste, and hair that mimics COVID illness. In addition, it causes dangerous hypercoagulability of blood that disrupts oxygen transport and delivery, causing sudden, unexpected death in some healthy young individuals and lingering "Long COVID" syndrome in others.

Introduction

This essay will argue that the exaggerated virulence of the "novel" coronavirus embodied in its "spike protein" causes the disparate pathologies of COVID, its congeners, and its immunizations by attacking the vascular endothelium, which induces harmful hyperactivity in the newly-discovered mammalian stress mechanism (MSM) [1]. Treatments that control MSM hyperactivity can reinstate health and save lives by restoring effective organ function.

Viral Virulence

Like other viruses that cause the "common cold," coronaviruses are obligate intracellular parasites that hijack lung cells to replicate themselves and induce coughing and sneezing that spreads their particulates to additional victims. Victim crowding exaggerates viral virulence, but killing victims constrains virulence. Variable viral virulence is not a mutation (an alteration of DNA), but rather an innate characteristic of the viral genome that is lacking in bacteria, and can be artificially exaggerated [2,3]. It explains why the common cold becomes more common during winter, when people are crowded together indoors, and why influenza killed more people than bullets and bombs during WWI after flu-stricken soldiers were quarantined in close quarter [4]. Variable virulence, however, does not explain the appearance of "novel" coronavirus contagions during peacetime, or why COVID and its congeners wax and wane unpredictably at random locations.

Conventional medical theory cannot explain the confusing symptoms and manifestations caused by the "novel" coronavirus, which caused the recent SARS, MERS, COVID-19, and OMICRON contagions. Some have speculated that autoimmune activity causes COVID pathologies, but there is no convincing evidence that immune activity harms tissues [5]. Others have speculated that a "neurological disease" causes the lingering muscle weakness, fatigue, and "brain fog" of the "Long COVID" syndrome suffered by survivors, but this doesn't explain the disparate forms of COVID mayhem including hypercoagulability, hypertension, heart attacks, strokes, pulmonary emboli, increased cancer, sudden death in healthy young individuals, stubborn bacterial infestations, anemia, and loss of hair, smell, and taste [5-9].

There is growing concern that mRNA COVID vaccines provoke sudden death in young, healthy people by propagating "spike proteins" throughout the body [10-24]. Most experts attribute these deaths to myocarditis and pericarditis that provokes "cytokine storm," fatal dysrhythmias, and "Commotio cordis" but this explanation seems weak for several reasons [21]:

1. Pericarditis and myocarditis cause warning symptoms of chest pain, fatigue, and malaise before they cause fatal dysrhythmias, but COVID-related sudden death occurs without warning symptoms [43, 44].
2. Myocarditis and pericarditis cause heart failure and hypotension rather than hypertension. [27].
3. Myocarditis and pericarditis resolve slowly, so they are inconsistent with the speedy recovery of victims of unexpected pulseless collapse who were successfully resuscitated by prompt CPR and defibrillation [45-47]
4. COVID-related sudden deaths resemble catecholamine injury rather than pericarditis and myocarditis at autopsy [48, 49].
5. Pericarditis and myocarditis don't readily explain stroke and pulmonary embolism [27, 30].
6. "Commotio cordis" (fatal spontaneous cardiac dysrhythmia) is a diagnosis of exclusion, when no other explanation is available.
7. MRNA vaccines cause systemic inflammation of the vascular endothelium that is not limited to the heart; it afflicts organs and tissues throughout the body, and it causes blood hypercoagulability and "Long COVID" symptoms cannot be explained by cardiac effects alone [6, 27, 31, 45, 50-56].

The "novel" coronavirus that caused human, chicken, and mink epidemics has much in common with the influenza virus that caused the devastating "Spanish Flu" epidemic in 1918:

1. Both viruses afflict euthermic mammals and birds.
2. Both can cause contagions.
3. Both afflict the pulmonary endothelium and cause life-threatening "adult respiratory distress syndrome" (ARDS); abnormal bleeding from intestines, eyes, ears, nose, and mouth; sudden death in young, healthy individuals; and lingering muscle weakness, fatigue, loss of hair, smell, taste, and other disabilities in survivors [4, 23, 24, 27, 28, 34, 38, 45, 50, 51, 57-79].

The WWI influenza deaths were often attributed to secondary bacterial infections, but bacterial effects cannot explain why so many young, healthy people died suddenly, sometimes within 12 hours of exposure why many bled abnormally; or why survivors suffered lingering muscle weakness, fatigue, and loss of hair, smell, hearing, and taste [80].

The similarities of COVID and epidemic influenza suggest that the same virulence characteristic causes the mayhem of these otherwise innocuous viruses. I hypothesize that this characteristic is embodied in the "spike protein" that is produced by the coronavirus and found in the tissues of COVID immunization victims [79]. I further hypothesize that this "spike protein" explains the variable virulence of the flu virus and other viruses that cause the "common cold" and influenza.

Stress Theory

Theories remain useless until they can be tested and confirmed, and powerful new theories typically arrive many years before evidence becomes available to confirm them [81]. For example, Miescher hypothesized that DNA is the medium of inheritance in the late 1800's, but his idea remained unverifiable until Watson and Crick discovered and described the DNA structure in 1953, which explains how DNA retains and replicates genetic information.

The DNA discovery focused attention on the "stress theory" of Dr. Hans Selye that postulates the presence of a "stress mechanism" that converts the DNA genetic blueprint into embryological development and then remains active throughout life to regulate tissue repair and organ function. Hyperactivity of the stress mechanism due to unremitting environmental stresses would explain the nature of disease, and its discovery would confer fresh treatments directed at its cause, and revolutionize medicine. Selye's ideas were and remain the best prospect of an effective theory of medicine, and they inspired an intense international search for the stress mechanism that lasted 30 years and consumed the careers of hundreds of researchers, the lives of thousands of tortured test animals, and millions (today billions) of dollars, but it failed to find any clue of the hypothetical stress mechanism, whereupon stress theory was relegated to the realm of the Unicorn and mostly forgotten. Another 30 years of accumulating information from unrelated research finally enabled its discovery by this unlikely amateur [81,82]. Thus discovered, the stress mechanism fulfills all the predictions and expectations of the old stress researchers [82]. It explains the nature of physiology, pathology, stress, and their relationships, and it provides a fresh, simplified, cohesive explanation of the confusing effects of COVID and influenza epidemics, as well as mRNA COVID vaccines.

The Mammalian Stress Mechanism (MSM)

The stress mechanism consists of the vascular endothelium, the nervous system, hepatic "coagulation" enzyme factors VII, VIII, IX, and X. The vascular endothelium is the focus of stress mechanism activity. It is a diaphanous layer of specialized cells, one cell thick, that lines the inner surface of all blood vessels and is the sole constituent of capillaries (see figure 1). It is therefore ubiquitous throughout the body, and it is sub-specialized to suit the requirements of various organs and tissues, which partially explains their differing reactions to stress (see figure 2).

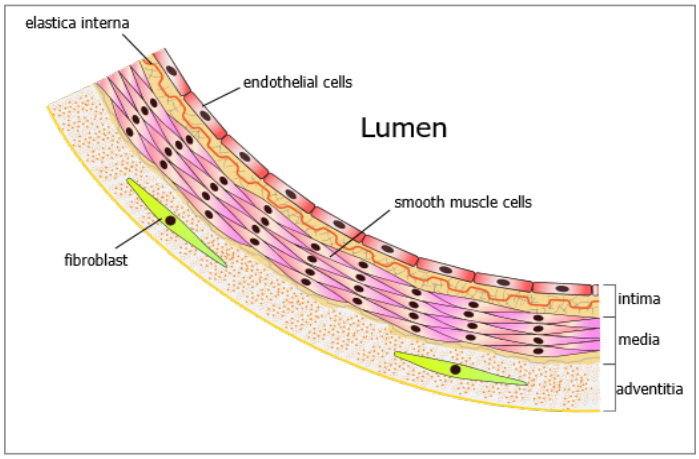

Figure 1: This drawing depicts the layer of vascular endothelial cells lining the inner surface of an artery. By en:User:StijnGhesquiere, user:Drsrisenthil - Complete Recreation and colorization based on the diagram originally produced by at File:Anatomy artery.png, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=58257331

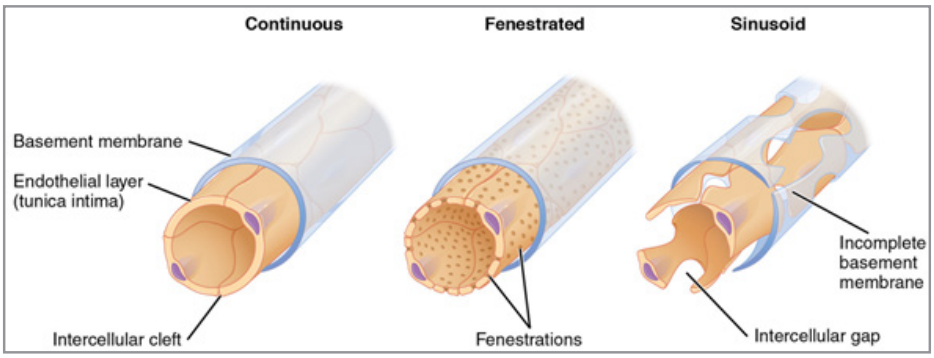

Figure 2: Capillary endothelium is specialized to serve the functions of organs and tissues. It is sinusoid in the liver to facilitate lipoprotein absorption, continuous in the brain to prevent the escape of tissue factor into systemic circulation, and fenestrated in muscle to foster nutrient uptake.

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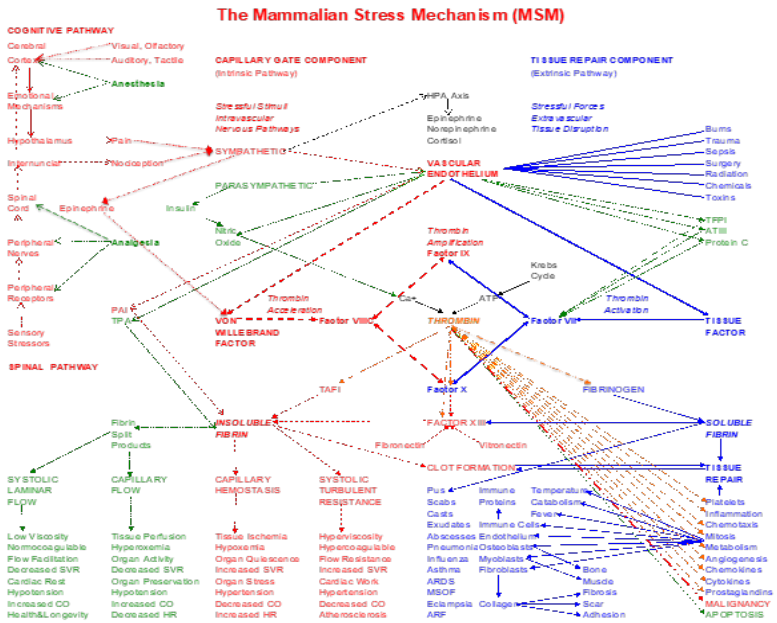

Figure 3: This diagram of the MSM illustrates the relationships of the vascular endothelium with blood enzymes, trauma, and nervous activity. Fluctuating factors VII and VIII alter the enzymatic interaction of factors VII, VIII, IX, and X to determine the

location, magnitude, and speed of production of thrombin, soluble fibrin, and insoluble fibrin to repair tissues and regulate organs. Their constant fluctuation focuses MSM effects that repair tissues and regulate organs, but fluctuating hyperactivity of the mechanism produces a bewildering blizzard of symptoms and manifestations that obscures its relatively simple operation.

The vascular endothelium performs several functions:

1. It isolates extravascular tissues from flowing blood, so that trauma initiates tissue repair by exposing blood enzymes to tissue factor in extravascular tissues.
2. It manufactures von Willebrand Factor (VWF) and releases it into flowing blood in accord with sympathetic nervous activity to "close" the capillary gate mechanism, which inhibits tissue perfusion and organ function.
3. Sympathetic nervous activity releases epinephrine from the adrenal glands, and epinephrine releases VWF from the vascular endothelium to close the capillary gate in muscles and tissues that lack direct sympathetic innervation.
4. It manufactures nitric oxide (NO) and releases it into blood in accord with parasympathetic nervous activity to "open" the capillary gate mechanism, which promotes tissue perfusion and organ function.
5. Parasympathetic nervous activity releases insulin from the pancreas, and insulin releases NO from the vascular endothelium to open the capillary gate in muscles and tissues that lack direct parasympathetic innervation.
6. It produces several other enzymes and substrates pertinent to stress mechanism operation including ATIII, fibronectin, vitronectin, and protein C.

The vascular endothelium orchestrates stress mechanism activity via two independently activated sub-components, each of which exaggerates the activity of the other via the enzymatic interaction of factors VII, VIII, IX, and X, which generates thrombin, soluble fibrin, and insoluble fibrin (see figure 3).

1. Thrombin is the "universal enzyme of extracellular energy." It enables extracellular cells and enzymes to use ATP energy to enable tissue repair and capillary gate activity. It energizes cell activities including metabolism, mitosis, chemotaxis, immune activity, and cell hormone release. Thrombin elevations cause inflammation, fever, malaise, and malignancy. Thrombin also energizes the conversion of fibrinogen to soluble fibrin, and the conversion of soluble fibrin to insoluble fibrin.
2. Fibrinogen is continuously produced by the liver and released into blood. It is the substrate of soluble fibrin and insoluble fibrin.
3. Soluble fibrin is the "universal substrate of body proteins" including insoluble fibrin, collagen, elastin, lipoproteins, mucus, saliva, and Mother's milk. When produced in excess it causes edema that disrupts organ function.
4. Insoluble fibrin is the "universal polymer of hemostasis." It enables coagulation, capillary hemostasis, scab formation, and capillary gate operation. When produced in excess it causes hypercoagulability of blood that invites infarction and amyloid production that causes rheumatoid diseases.

Combinations of fluctuating autonomic balance and tissue damage determine how and where the MSM generates thrombin, soluble fibrin, and insoluble fibrin. The constant fluctuation of the sub-mechanisms focuses stress mechanism effects and generates "positive feedback" to repair tissues and regulate organs:

1. The tissue repair component repairs tissues in accord with tissue damage. Surgery, sepsis, trauma, burns, radiation, toxic chemicals, viruses, and so forth disrupt the vascular endothelium and expose tissue factor in extravascular tissues to factor VII in flowing blood. This activates factor VII enzyme activity in a dose-related manner, which causes the enzymatic interaction of factors VII, VIII, IX, and X to generate thrombin that energizes tissue repair activities including hemostasis, inflammation, chemotaxis, mitosis, metabolism, immune activity, cell hormone release, cell differentiation, and angiogenesis [83].
2. The Capillary Gate Component governs microvascular flow resistance in accord with autonomic balance, which fluctuates in accord with all forms of nervous activity. Sympathetic nervous activity releases VWF from the vascular endothelium into blood to close the capillary gate, and parasympathetic nervous activity releases nitric oxide (NO) from the vascular endothelium to open it. Carbon dioxide regulates oxygen transport and delivery by directly releasing NO from the vascular endothelium to open the capillary gate [84]. Autonomic balance thus regulates organ perfusion, which governs organ activity [85].

The stress mechanism repairs tissues and regulates organs efficiently and unobtrusively, but like any mechanism, it has its limits. Excessive and unremitting combinations nervous stimulation and tissue disruption can induce MSM hyperactivity. This causes the mechanism to waste its substrates and produce harmful excesses and defective versions of thrombin, soluble fibrin, and insoluble fibrin. Such stress mechanism hyperactivity manifests as disease.

Tissue Specialization Affects Stress Mechanism Activity

The lungs, brain, retina, nerves, arteries, gonads, placenta, and cervix are rich in tissue factor [86]. This amplifies tissue repair activity, which exaggerates the vulnerability of these "target tissues" to stress and cancer.

The vascular endothelium of internal organs, especially in lung, brain, and bowel, is directly innervated by autonomic nerves that regulate the capillary gate mechanism in accord with autonomic balance [82]. This exaggerates the sensitivity of internal organs to stress.

The lungs and brain are especially vulnerable to stress and malignancy, because they are rich in both tissue factor and direct autonomic innervation.

"The lungs are uniquely vulnerable to viral attack due to their specialized structure and direct exposure to airborne pathogens. Lungs are composed of alveoli, which consist of a single layer of pneumocytes that are surrounded by specialized capillaries that bathe their outer surface in blood. The capillaries consist of a single layer of endothelial cells whose basement membrane fuses with that of the pneumocytes [82,87]. This delicate, thin interface optimizes gas exchange in the lung [84]. The cells of the vascular endothelium are devoid of tissue factor, but the pneumocytes are rich in tissue factor, so that viral disruption of this delicate cellular interface readily leaks tissue factor into

systemic circulation, which harmfully activates factor VII and exaggerates thrombin generation."

More detailed and fully-referenced explanations of the stress mechanism and its implications, and detailed explanations of blood turbulence and DIC can be found in my published papers, which can be downloaded from my website www.stressmechanism.com, or in my book "50 Years Lost in Medical Advance: The Discovery of Hans Selye's Stress Mechanism" that is available via Amazon.com [82,85,88-94,1].

Normal Coronavirus and the Common Cold

The ordinary coronavirus produces "spike protein" that enables it to invade cells, where it hijacks cell machinery to replicate itself. This causes an inflammatory reaction that increases the permeability of lung tissues and allows increased "penetration" of factor VII in flowing blood into lung tissues. Tissue factor in lung tissue binds to the factor VII, which increases thrombin generation. The increased thrombin generation energizes immune activity, exaggerates inflammation, and induces pulmonary exudate (mucus) production. This causes coughing and sneezing, which produces clouds of viral particulates that spread the infection to additional victims until the increased mucus production and immune activity rids the virus from the body. However, the generalized immune activity induced by the increased stress mechanism activity does not confer specific immunity to the virus.

The pulmonary inflammation also increases the "leakage" of tissue factor from lung tissues into blood circulation, where it interacts with factor VII and exaggerates thrombin generation via the interaction of factors VII, VIII, IX, and X, which exaggerates cellular metabolism throughout the body. This wastefully converts ATP energy into heat. These effects explain the fever, fatigue, and malaise caused by the common cold. However, these symptoms are normally mild and self-limiting, and cause neither lasting harm in healthy individuals nor specific immunity to the virus.

The "Novel" Coronavirus, COVID, and Critical Illness

The "novel" coronavirus produces a more virulent version of the spike protein than normal coronavirus. It thus causes greater inflammation, immune activity, exudate production, and leakage of tissue factor into systemic circulation than the normal coronavirus. The resulting severe increase in stress mechanism hyperactivity manifests as "viral pneumonia" or "adult respiratory distress syndrome" (ARDS), which is a life threatening "critical illness" because it produces excessive pulmonary exudates that disrupt gas exchange and promote pulmonary sclerosis, and causes severe systemic inflammation that progressively disrupts organ function. This manifests as a life-threatening "critical illness."

Critical Illness

Combinations of excessive sympathetic nervous activity that releases von Willebrand Factor from the vascular endothelium and excessive permeability of the vascular endothelium can cause severe, generalized hyperactivity of the stress mechanism that disrupts organ function, threatens survival, and manifests as "critical illness. "This occurs in a wide variety of stressful circumstances, including major trauma, sepsis (systemic bacterial infestation), excessive radiation, pneumonia, extensive surgery, poison exposure, severe burns, pregnancy, cardiac bypass, and so forth. Conventional medical theory cannot explain critical illness, and can only describe "syndromes" (groups of symptoms that collectively indicate or characterize a disease). For example, critical illness that occurs during pregnancy is called "eclampsia." The "Surgical Stress Syndrome" (SSS) occurs when general anesthesia is inadequately supplemented with analgesia during surgery. "Systemic Inflammatory Response Syndrome" (SIRS) occurs when patients subjected to coronary artery bypass during anesthesia are not adequately supplemented with narcotics. These various syndromes are typically confused with one another because they are all essentially the same.

Chronic Illness

I hypothesize that persistent, sub-clinical stress mechanism hyperactivity that exaggerates "fibrin turnover" that elevates the generation of abnormal amyloid protein. The amyloid accumulates in various tissues and induces chronic inflammation that inexorably accelerates capillary senescence, tissue oxygen starvation, collagen generation, and tissue sclerosis that damages tissue function. This manifests and chronic illnesses called "rheumatoid disease" that manifests as diabetes, hypertension, atherosclerosis, congestive heart failure, Systemic Lupus Erythematosus (SLE), scleroderma, Sjogren's disease, and so forth. Obesity causes chronic stress mechanism hyperactivity that manifests as chronic illness.

Patients who suffer from obesity or pre-existing chronic illnesses ("rheumatoid" diseases) are prone to develop "critical illness" when they are afflicted by the novel coronavirus. This is because chronic illness compromises oxygen transport and delivery, and undermines organ function [84]. These harmful effects exaggerate the morbidity and mortality of the "novel" corona virus.

The effects of COVID pneumonia are not limited to the lung, because the lung is rich in tissue factor and autonomic innervation. COVID pneumonia thus releases copious quantities of tissue factor from the lung into systemic circulation, where it harmfully elevates thrombin generation throughout the body, causing systemic inflammation. Thrombin converts fibrinogen into soluble fibrin that penetrates through the inflamed vascular endothelium into organs and tissues, where it causes tissue edema that disrupts organ function. This progressive is called "Multi-Organ Failure Syndrome" (MOFS). The brain is the next organ to be afflicted, because like the lung it is rich in both tissue factor and autonomic innervation. Brain inflammation manifests as delirium and dementia that culminates in coma, and it releases still more tissue factor into systemic circulation.

The increasing MSM hyperactivity disrupts the glycocalyx, which is a delicate molecular mesh produced by the vascular endothelium that lines the interior surface of all blood vessels. [95-97] This releases albumen protein from the glycocalyx into flowing blood, where it is removed by the kidneys and appears in the urine as "albuminuria." This serves as an early warning of impending stress mechanism hyperactivity. Soluble fibrin is also removed from blood into urine and is called "proteinuria." Albumen and soluble fibrin clog renal tubules and halt urine production. This is called "Acute Renal Failure" (ARF). If the condition persists, it causes permanent kidney damage called "Chronic Renal Failure" (CRF) [95,97-99].

Soluble fibrin invades the skin, causing visible edema. It likewise invades organs and disrupts their function. This sometimes causes the liver to swell and burst [100]. It also invades bowel tissue, which halts peristalsis. This is called "bowel ileus."

Thrombin inflammation sensitizes nociceptors, which elevates sympathetic nervous activity that releases VWF from the vascular endothelium. This increases factor VIII activity, which increases insoluble fibrin production that elevates blood coagulability. This invites infarction and DIC (disseminated intravascular coagulation).

Meanwhile, rising blood levels of insoluble fibrin exaggerate blood viscosity (microvascular flow resistance) and coagulability. This inhibits the ability of the turbulent pulse waves to maintain arterial patency by preventing clot formation, and promotes the risk of myocardial infarction, stroke, pulmonary embolus, and DIC (disseminated intravascular coagulation).

Disseminated Intravascular Coagulation (DIC)

Disseminated Intravascular Coagulation (DIC) is sudden, spontaneous arterial coagulation combined with unexplained bleeding from the mouth, intestine, eyes, and so forth. It is clinically confusing, because coagulation involves a fluctuating balance of factors that promote and prevent coagulation. The least appreciated of these factors is pulsatile blood turbulence. Each cardiac contraction generates a wave of pulsatile arterial turbulence that propagates throughout the arterial tree during diastole, momentarily halting blood flow as it travels. Its intensity increases with decreasing arterial diameter. It maintains arterial patency by inhibiting atherosclerosis and clot formation, and disintegrating intra-arterial clots that obstruct arterial flow [88, 89, 92, 94].

Red cell mass affects arterial turbulence. The small size, biconcave shape, and neutral buoyancy of mammalian red blood cells causes them to spontaneously form "Rouleau" or "stacks" that suppress turbulent flow resistance during systole. This enhances mammalian exercise tolerance, but if red cell mass becomes excessive, it can inhibit arterial turbulence to the point that DIC begins spontaneously. For example, overenthusiastic treatment with Epogen, which is artificial erythropoietin that is used to treat anemia, can dangerously elevate red cell mass and cause lethal DIC [8, 101]. Another example is "blood doping" by athletes who dose themselves (illegally) with Epogen in order to optimize their exercise tolerance and athletic performance [102]. This is a dangerous practice and has caused DIC deaths [103].

On the other hand, severe anemia, such as occurs during severe chronic renal failure, can exaggerate pulsatile blood turbulence to the point that hemostasis fails, and uncontrolled bleeding begins. [104,105]. The exaggerated turbulence also increases blood viscosity (flow resistance), which impedes oxygen transport and delivery to tissues, and causes oxygen starvation. This explains why severe anemia causes chronic fatigue, weakness, and poor exercise tolerance. [105,106]. This can be treated with transfusions of packed red blood cells that reduce turbulence and restore effective coagulation.

Yet another factor that affects blood coagulation is MSM hyperactivity that elevates insoluble fibrin generation in blood [82,94]. Insoluble fibrin exaggerates blood coagulability, inhibits pulsatile turbulence, and binds red cells together into clots [89].

DIC begins in small peripheral arteries, where turbulence declines exponentially with increasing coagulability. If it proceeds rapidly, it can disrupt oxygen transport and delivery, causing sudden death. More often, it consumes, wastes, and depletes coagulation substrates including fibrinogen, fibronectin, and vitronectin, and forms defective clots that remove red cells from circulation and deposits them on the inner walls of arteries. The resulting anemia exaggerates turbulence, which increases flow resistance and inhibits coagulation [105,106].

This provides a brief introduction to DIC details pertinent to this presentation. Those who seek a more thorough and fully referenced explanation of this complex subject can find it in my book [1].

COVID Immunizations, Sudden Death, and Long COVID Syndrome

The COVID mRNA vaccines are more dangerous than COVID itself because the mRNA "vaccine" introduces what amounts to live virus directly into systemic circulation, where they cause cells to propagate harmful "spike protein" throughout the body [50,107-110,61]. This causes the vascular endothelium to release VWF into systemic circulation, which activates factor VIII that generates insoluble fibrin, and dangerously exaggerates blood coagulability.

In older patients, whose microvascular perfusion is compromised by capillary senescence, the rising blood coagulability elevates the risk of myocardial infarction, stroke, thrombosis, and congestive heart failure [09,111]. The DIC causes microthrombi that clog capillaries, and abnormal calamari-like strands of insoluble fibrin that occlude small peripheral arteries, obstruct cardiac output, disrupt oxygen transport and delivery, and persist after death [69]. However, the calamari strands are typically discovered during embalming rather than autopsy, because conventional autopsy focuses on large central arteries rather than small peripheral arteries.

Healthy young people develop a more vigorous stress response to COVID and influenza epidemics than elderly victims, which explains why the victims of sudden death are predominantly young and healthy [25,42,109,112]. In younger patients, whose microvascular circulation remains intact, the increasing blood coagulability doesn't cause warning symptoms until it rises above a critical threshold, whereupon it suddenly and unexpectedly triggers a unique form of disseminated intravascular coagulation (DIC) that has yet to be recognized. This causes brain hypoxia, loss of consciousness, and sudden death. The deaths are painless, because the brain cannot detect pain in its own tissues, and it quickly depletes its supply of oxygen, which extinguishes consciousness before painful hypoxia develops in other organs and tissues. This happens so quickly that the calamari strands do not develop, because plasmin activation disintegrates the causative clots soon after death, leaving only the inflamed vascular endothelium as evidence. As a result, most DIC deaths in healthy young people are attributed to myocarditis pericarditis, cardiac tamponade, or congestive heart failure, but this is illogical, because these conditions typically cause warning signs of fatigue and malaise before causing death [113]. Instead, the autopsies report that the heart is distended, as if the patients died of catecholamine overdose, but this cannot be explained

by conventional theory [48,52,114]. This cardiac distention is explained by the sudden obstruction of cardiac output due to DIC that obstructs small peripheral arteries and capillaries [17,20,33,43,45,52,65,66,68,69,79,115-119]. The phenomenon appears to be fleeting, because some victims have been successfully resuscitated when trained CPR technicians and defibrillation equipment are immediately available. Such survivors sometimes exhibit abnormal unexplained bleeding in lungs and body orifices [45,46, 118,119]. This is consistent with spontaneous clot disintegration due to plasmin activation.

DIC is not invariably lethal. Sometimes the DIC process removes red cells from circulation, causing anemia that exaggerates pulsatile turbulence, which halts the clotting process before it becomes fatal. However, the abnormal coagulation clogs small arteries and capillaries, and causes anemia that exaggerates turbulent flow resistance, as in patients with the anemia of chronic renal failure. The increased flow resistance inhibits oxygen transport and delivery that manifests as fatigue, muscle weakness, and mental "fog." The small renal arteries are especially vulnerable to clogging by the abnormal coagulation, which explains why many patients suffer intense hypoxic renal pain after COVID injections. The abnormal coagulation may also cause lasting damage to the arterial tree and capillaries that undermines oxygen transport and delivery, which explains persisting muscle weakness, fatigue, mental "fog" and vulnerability to stubborn infections that is called "Long COVID."

Meanwhile, as the propagating spike protein attacks the vascular endothelium throughout the body, it causes pericarditis, myocarditis, pericardial effusions, cardiac tamponade, miscarriages, testicular edema, infertility, malaise, lingering loss of hair, smell, and taste, and stubborn infections [18,31,40,43,117,120,121]. Furthermore, the lingering MSM hyperactivity exaggerates the risk and severity of cancer, heart disease, and chronic illnesses. [31,57-61,71,75, 115,121,122]. In some patients these symptoms and conditions persist for months or even years, and they account for the "Long COVID syndrome" that is caused by COVID immunizations.

Pre-existing Illness and COVID

Pre-existing chronic illnesses exaggerate the severity of COVID and influenza contagions. Chronic illnesses have been increasing exponentially for years on account of environmental pollution [123]. They include seemingly unrelated conditions including diabetes (type I and type II), hypertension, atherosclerosis, systemic lupus erythematosus (SLE), Sjogren's Disease, scleroderma, Parkinson's Disease, Alzheimer's disease, polymyalgia rheumatica, rheumatoid arthritis, gout, and multiple sclerosis. Conventional medicine classifies these illnesses as "rheumatoid diseases" or "autoimmune" diseases. However, there is no convincing evidence that immune activity per se causes them.

Many chronic illnesses are closely associated with specific forms of environmental stress. For example, obesity is closely associated with atherosclerosis, hypertension, and diabetes; SLE is associated with cosmetics exposure; and Parkinson's disease is closely associated with pesticide exposure. Conventional medical theory cannot explain these observations.

The cause of chronic illnesses supposedly remains unknown, but the research literature reveals amyloid protein accumulation in the afflicted tissues of all rheumatoid diseases. Conventional medicine ignores this and views amyloidosis as a rare, mysterious, and unrelated disease. The MSM explains amyloidosis. Unrelenting environmental stresses such as pesticides, herbicides, automobile exhaust, chlorinated water, and industrial pollution increasingly contaminate the beverages we drink, the food we eat, and the air we breathe. To this may be added the emotional adversity of difficult marriages, frustrating jobs, and the eternal injustice of government justice, all of which induce stressful nervous activity that harmfully stimulates the MSM. These stresses persistently exaggerate the generation of insoluble fibrin and its subsequent disintegration into "fibrin split products" (FSP) [124]. The FSP is normally harmless and is re-cycled by the body. However, some of the FSP re-interacts with coagulation factor X to produce abnormal "amyloid" protein that accumulates in organs and tissues. Thus, environmental stress exaggerates insoluble fibrin "turnover" and amyloid production. As a result, atherosclerosis and amyloidosis occur in concert, and substantial amyloid accumulation appears in the abnormal clots and strands of insoluble fibrin caused by DIC [125]. The amyloid induces subclinical inflammation and immune activity in the afflicted tissues of rheumatoid diseases. It accelerates capillary senescence, which undermines tissue oxygenation and promotes pathological collagen generation that causes debilitating sclerosis in organs and tissues [126-129]. Such pre-existing organ and tissue damage exaggerate morbidity and mortality in COVID victims.

Clinical Examples

Though critical details are usually lacking, the following examples gleaned from public reports illustrate how COVID and its immunizations cause heart attacks, strokes, blood clots, DIC, unexplained sudden death in healthy young people, and "Long COVID" syndrome.

Example # 1
Susan D

Although I am a board-certified anesthesiologist with more than 40 years of experience, I failed to notice the controversial deaths and "Long COVID" problems associated with the immunizations until my chance acquaintance with Susan D., a previously healthy 75 y.o. school psychologist who was obliged to undergo five Modern COVID-19 immunizations over a period of 18 months on penalty of losing her job.

Susan's first four immunizations caused no distress, but her fifth immunization on 9/15/2022 was a "bi-valent booster" which caused sharp pain during the injection. About three minutes later this was followed by weakness, fatigue, fatigue, mental fog, malaise, nausea, chills, and dizziness. The sudden onset of weakness, mental fog, and fatigue is best explained by sudden MSM hyperactivity that triggered a non-lethal episode of DIC that impaired oxygen transport and delivery. Despite these severe symptoms, she drove herself a short distance home, and went to bed. On the following day, 9/16/22, she experienced flu-like symptoms including nausea, weakness, chills, and fatigue. That night she suffered sudden, searing pain in her right flank that was exaggerated by touch (allodynia). The most likely explanation abnormal DIC coagulation that obstructed blood flow to her right kidney and caused hypoxic kidney pain [130]. The next morning, 9/17/22, she visited an emergency room, where she was treated with Toradol 30 mg IM to control the pain. A com-

plete blood count (CBC) revealed that her hemoglobin level was 6.5 gm/dL. (half normal). Because of this, she was transfused with a unit of blood andiron pills were prescribed even though a rectal examination revealed no blood and she had no history of black tarry stools or other evidence of blood loss. The most likely cause of this sudden, unexplained onset of severe anemia is DIC that sharply reduced circulating red cell mass. A kidney stone was suspected and she underwent a CAT scan of her entire body, but the results were negative. That night, 9/18/22, the flank pain resumed and she returned to the emergency room, where she was treated with another 30mg Toradol IM. This controlled the pain and it never returned. The simplest explanation is that the anticoagulant effects of the Toradol together with increased pulsatile turbulence of anemia disintegrated the blood clot that obstructed perfusion in her right renal artery. A repeat CBC on that occasion showed that the transfusion had raised her hemoglobin to 7.9 gm/dL, which was still low. She continued to suffer fatigue, weakness, exercise intolerance, and mental fog. This reflects the generalized tissue hypoxia she suffered due to her near-lethal episode of DIC that compromised oxygen transport and delivery.

On 10/10/22 she underwent fiberoptic examination of her upper and lower GI tract, which failed to find any evidence of GI bleeding. This confirms my hypothesis that her anemia was caused by DIC. Her doctors, as well as I, were baffled by her lack of trauma, surgery, hematuria, tarry stools, bright red blood in her stools, or any other explanation of her anemia. On 11/27/22, approximately two months after the problematic COVID immunization, she suffered fresh symptoms of COVID, including worsened fatigue, sore throat, a dry cough, and atelectasis. COVID was confirmed by testing on 11/30/22.This is consistent with reports that the COVID immunizations actually cause COVID. By that time her hemoglobin level had risen to 10 gm/dL. This suggests that the abnormal DIC clots had spontaneously disintegrated due to plasmin generation, and the red cells had been re-mobilized by pulsatile blood turbulence that normally maintains arterial patency.131A subsequent COVID test on 12/11/22 was negative, but as of 12/17/22 she continued to complain of sore throat, shallow cough, headache, atelectasis, fatigue, poor exercise tolerance, and brain fog. Similar symptoms are widely-reported characteristics of the "Long COVID" syndrome [57,58,74,75,132-134].

Susan's symptoms slowly improved, and by Tuesday, 1/10/23 her hemoglobin had reached 11.8 gm/dL, which is essentially normal, and she had returned to using her treadmill machine, and no longer complained of "brain fog." On 1/12/23 she underwent repeat endoscopy to remove a colon polyp discovered by the previous endoscopy, but this second endoscopy also failed to reveal any source of gastrointestinal bleeding.

I remained as puzzled by Susan's fatigue and unexplained anemia as her doctors until I discovered a YouTube interview with Dr. Jaco Laubscher, a South African internist who observed that "Long COVID" patients, like DIC victims, exhibit damaged vascular endothelium, increased peripheral blood flow resistance, and hypercoagulable blood, and that anticoagulants relieve their symptoms" [51,59]. With this fresh information, plus my understanding of stress theory, I surmised that Susan had suffered an episode of DIC, which explained her unexpected anemia and symptoms of fatigue, muscle weakness, renal pain, and mental fog. Since then, accumulating reports have corroborated my suspicions [32,33,59,115].

What is unique about Susan's experience is that her CBC test was drawn at a critical moment that revealed the severity of her unexplained anemia. This clue is lacking in most reports. Furthermore, she didn't exhibit the abnormal bleeding, edema, and skin lesions that typify DIC.

The cause of Susan's injection site pain and immediate malaise remains unclear, but it invites the following possibilities:

1. Her previous COVID immunizations may have exaggerated sensitivity to the vaccine that caused the fifth immunization to be painful.
2. The needle might have struck a nerve or periosteum (the innervated sheath surrounding bone)
3. The vaccine may have been accidentally injected into a vein, so that it was quickly distributed systemically.

Susan's sudden symptoms of weakness, fatigue, dizziness, mental fog, nausea, and half normal CBC test immediately following her 5th COVID booster are best explained by DIC that trapped half of her circulating red cell mass in defective clots that were deposited on the inner walls of her arteries. Like the anemia of chronic renal failure, the DIC reduced the circulating mass of red cells, which exaggerated turbulent flow resistance, undermined oxygen transport and delivery, and caused her sudden symptoms of fatigue, weakness, dizziness, nausea, and brain fog. Her right flank pain was most simply explained by abnormal clot formation that obstructed her right renal artery [54]. Fortunately, the clot was soon cleared by pulsatile turbulence, which restored perfusion and oxygenation to the kidney, and eliminated the hypoxic kidney pain. As of this date (1/24/2023) there is no evidence of residual renal damage, and her CBC tests are now normal. She still suffers from residual loss of smell, taste, and hair, but has recovered her muscle strength and mental clarity, and she has resumed her normal activities including daily exercise on her treadmill. In my opinion, she is fortunate to have survived without more serious sequelae.

I recommended that Susan obtain a thromboelastographic (TEG) or d-Dimer test to assess her blood coagulability and residual risk for continuing MSM hyperactivity, but the "gatekeeper" (family physician) of her medical plan categorically refused consent.

Example #2
Charlbi Dean [66,67]
Charlbi Dean was a healthy 32 y.o. actress who awoke from sleep with the perception of medical distress during the night and alerted her fiancé to take her to the hospital, where she died in his company. She did not complain of pain. I hypothesize that she was awakened by a poorly understood protective brain mechanism that detects hypoxia during sleep and restores consciousness during "obstructive sleep apnea" where the afflicted person repeatedly becomes hypoxic and regains partial consciousness on account of airway obstruction that re-occurs with the return of sleep. Unfortunately, the restoration of consciousness didn't disrupt the DIC.

The New York Medical Examiner attributed her death to her previous history of splenectomy that supposedly caused an obscure form of bacterial sepsis, even though her autopsy revealed evidence of a viral infection in her lungs that may have been COVID. This is a weak explanation given the absence of any evidence of illness before her sudden death.

Her immunization history was not made public, but her sudden death is most simply explained by DIC secondary to COVID or COVID immunization, even though persons suspicious of her cause of death were disparaged as "anti-vaxxers" [66,67]. Unfortunately, no blood test results, if any, were revealed to the public, but the limited evidence available suggests that the DIC phenomenon doesn't necessarily cause immediate death, and that she could have been saved by timely treatment with anticoagulant therapy if her critical condition had been understood.

Example #3
Hunter Brown
Hunter Brown was a healthy 21-year-old cadet at the American Air Force Academy and a valued member of the academy football team. He was undoubtedly immunized for COVID because this is a military requirement [68]. He collapsed while walking to class, but there was no ambulance or defibrillation equipment available and CPR failed to save him.

Example #4
As Reported by Debbie Kulick
A healthy young woman was walking to a football game with her husband, a trained paramedic, when she suddenly complained that she didn't feel well, and then collapsed in cardiac arrest to the blacktop in a parking lot [46]. Her husband noted that she did not have a pulse, and immediately called for help and began CPR. Athletic trainers, a doctor, and an ambulance with defibrillation equipment were immediately available. She regained consciousness and resumed breathing after she was defibrillated using an AED, and was transported to a hospital. The simplest explanation is that DIC occurred secondary to COVID immunization, soon followed by spontaneous plasmin disintegration of abnormal DIC blood clots that enabled successful CPR and defibrillation that restored oxygen transport and delivery [84]. Unfortunately, no revealing blood tests, if any, were made available to the public. As in the case of Charlbi Dean, she remained conscious long enough to realize that she was dying, but did not complain of pain. This is consistent with present understanding that brain viability lingers for several minutes despite oxygen starvation.

Example #5
Autopsies of Two Adolescents Who Died After COVID Vaccination [48]

Autopsy Results
The microscopic examination revealed features resembling a catecholamine-induced injury, not typical myocarditis pathology.

Conclusions
The myocardial injury seen in these postvaccine hearts is different from typical myocarditis and has an appearance most closely resembling a catecholamine-mediated stress (toxic) cardiomyopathy. Understanding that these instances are different from typical myocarditis and that cytokine storm has a known feedback loop with catecholamines may help guide screening and therapy.

These autopsy results are consistent with the hypothesis that DIC suddenly exaggerates arterial flow resistance in the manner of catecholamine injury, halts cardiac function, and disrupts oxygen transport and delivery. This, plus COVID myocarditis, would explain the cardiac enlargement and cardiomyopathy observed at autopsy.

Example #6
Caddie Collapses, Given CPR During AT&T Pebble Beach Pro-Am Golf Tournament [119]
"From my perspective, it seemed like we lost him," Nelson said. "Luckily there was a police officer on the sidelines there and he [performed] CPR. So, he came in and effectively saved his life."

Higgs said he was then informed the caddie's condition had improved and that he would be OK.

This incident is interesting because the victim survived without the need for defibrillation, which suggests that spontaneous plasmin disintegration of the defective clots must have enabled successful CPR resuscitation.

Example #7
Yasmin Vossoughian open up about Health Scare
Yasmin is a news anchor on national television. Her illness illustrates how viral pericarditis, pericardial tamponade, and myocarditis cause chest pain, dyspnea, fever, fatigue, and other warning symptoms [43]. Her cardiologist arbitrarily attributed the illness to "autoimmune activity" but immune activity has never been demonstrated to cause disease. Her pericarditis, which caused the cardiac tamponade, was most likely caused by COVID vaccination. Unfortunately, her vaccination history was not revealed.

Example #8
Princess of Thailand Collapsed after COVID jab [116, 135].
The healthy 40 y.o. daughter of the king of Thailand collapsed suddenly 23 days after her third COVID immunization and remains in a coma. A world-famous expert claim that the COVID immunization caused her collapse. The king is reportedly furious.

Example #9
Elon Musk felt like he 'was dying' after 2nd COVID booster shot, cousin in 'peak health' suffered myocarditis [29].

Like Susan, Elon Musk experienced injection site pain, COVID symptoms, and a severe reaction that lasted several days after multiple COVID immunizations and boosters required by his work. He also stated that his cousin, who was in excellent health, suffered cardiac myositis that required hospitalization after he was immunized. He has not provided further details

Airline Pilots
Both military and commercial airline pilots have always been subject to strict medical standards. It is thus highly unusual for

any active pilot to suffer an unexpected medical emergency [136]. However, soon after COVID vaccination became mandatory, pilot Bob Snow suffered a heart attack six minutes after landing his airplane carrying 200 passengers [137]. The incident was not investigated by either the airline or the FAA, but soon afterward the FAA loosened EKG standards for pilots, which suggests that cardiac problems have increased since COVID immunizations became mandatory [55, 56, 138]. Perhaps even more disturbing is reports that "co-pilots" may no longer be present on some flights [139]. Here are some reports from the month of March, 2023:

Example #1
Southwest Boeing 737
On March 22, 2023 a Southwest Airlines flight from Las Vegas (Nevada) to Columbus (Ohio), saw its captain become incapacitated. The Aviation Herald notes that the Boeing 737-700 was end route at FL370 about 160nm northeast of Las Vegas when the first officer radioed air traffic control to notify them that the captain was complaining about stomach pain. It was then noted that the captain then became incapacitated. In a similar resolution to the Air Transat situation, another pilot was available to step in. In this case, it was a passenger that was an off-duty, fully-licensed pilot for a different airline [140].

Example #2
On March 18, 2023 an Air Transat Airbus A321 was flying from Fort-de-France to Montreal when the aircraft's first officer became incapacitated. The incident occurred as the aircraft was flying over the United States, 200NM south of Montreal. According to The Aviation Herald, the incident took place aboard Air Transat flight TS739, an Airbus A321-200 service from Fort-de-France, capital of the French territory of Martinique to Montreal, Canada. The flight departed took off at 13:34, 34 minutes past its scheduled departure time. According to FlightRadar24.com data, reached a cruising altitude of FL320 approximately 25 minutes after takeoff. The aircraft increased its cruising altitude twice more during the flight, going up to FL340, and then FL360. 191 people were on board the A321-200, which was registered C-GTCY [140].

Despite two such instances taking place within a week, these cases are thankfully quite rare. However, the top priority for aviation is safety. Thus, the reality of the occasional "incapacitated pilot" incident is one strong argument against the possibility of single pilot operations. Two pilots in the cockpit provide redundancy so that if one of the aircraft's pilots were to become incapacitated, the other pilot could take over the full operation of the aircraft and perform a landing.

Last month, a Reuters report noted that the European Union Aviation Safety Agency (EASA) had ruled out single-pilot flights by 2030. However, the regulator was reportedly still evaluating proposals from Airbus and Dassault Aviation. This could see limited single-pilot operation for parts of the flight as early as 2027.

It would appear, however, that some proposals for single-pilot operations would still include at least one other pilot onboard the aircraft. This is the case for Airbus and Cathay Pacific's 'Project Connect', which envisions a fatigued pilot being able to rest in the crew rest area while the other pilot flies' solo during less-demanding portions of the flight, and not takeoff or landing [140].

Example #3
March 13, 2023 Emirates Flight EK205 MXP-JFK from Milan diverted on March.13, 2023 due to pilot illness, returned to Milan for emergency landing - now 4th pilot incident this month. Dubai: Emirates cancels flight, returns to origin destination after take-off [141].

Example #4
United Airlines Flight 2007 GUA-ORD from Guatemala to Chicago diverted on March 11, 2023 due to "incapacitated pilot" who had chest pains - now 3rd pilot incident this month.

A United Airlines flight from Guatemala to Chicago was diverted to Houston's George Bush International Airport on Saturday evening. An emergency was declared "for an incapacitated pilot," stated a Houston UAL internal document given to CDM Press. "UA Flight 2007 GUA-ORD is diverting to IAH. Declaring an emergency for an incapacitated pilot. Gated at E20. Current ETA shows 1747. Unknown if flight will clear here at this time or just re-crew and go. Will advise when information is available. Pilot reportedly taken to a hospital," states the UAL Operation Center communication. According to the UAL file, "Left seat Capt. had chest pains. Could not get him out of the seat. Right seater landed. "On Saturday evening, KHOU-TV Houston reported that the airport spokesperson told that news organization that the flight was diverted "due to a technical issue." UAL's CEO Scott Kirby has prided himself for making mandatory COVID vaccinations part of UAL's branding since August 2021 when he first mandated the shots.

At that time, UAL employees could apply for a religious or medical exemption. Kirby has claimed publicly that 99% of their employees have been vaccinated. But, there is more to Kirby's proclamations. If a religious exemption was granted, employees were put on "unpaid leave." They did not have access to any of their benefits, including medical, vision, life, and their 401(K) accounts. If they wanted to continue with their medical insurance, they had to make payments which was burdensome because they were not receiving paychecks. It was only in March 2022 when those put on unpaid leave were invited back to work [141].

Example #5
March 11, 2023 BA TRAGEDY Veteran British Airways pilot collapses and dies shortly before he was due to captain packed passenger jet [142].

Example #6
Virgin Australia flight from Adelaide to Perth forced to make emergency landing as First Officer suffered heart attack 30 minutes after departure on March 3, 2023. Virgin Australia Pilot Suffers 'Heart Attack' 30 Minutes After Takeoff, Prompting Emergency Landing [143].

NFL Football Players [136]
95% of all NFL football players are known to be COVID vaccinated as a requirement of their jobs, and a surprising number of these healthy young men have died suddenly without explanation or suffered coagulation abnormalities, strokes, and heart attacks since the appearance of COVID and its immunizations. Blood doping doesn't explain these problems, because that practice became commonplace among professional athletes long be-

fore COVID, and its dangers are well known [8, 103]. Medical information that might clarify what happened in these cases, such as tests of blood coagulability, CBC results and so forth, are not made available to the public, but unexplained deaths, blood clots, heart attacks, and strokes in healthy young men at any age are rare and noteworthy. The increase in such problems in NFL players since the onset of the COVID contagion is best explained by hypercoagulability of blood secondary to COVID immunizations.

Example #1
Damar Hamlin [45-47, 118] was 24 years old when he suddenly collapsed during a nationally televised football game, and he would surely have died were it not for the prompt medical attention he received. He was defibrillated twice on the scene, and then intubated and transported to a nearby hospital, where he was placed in intensive care. Abnormal bleeding from his lungs was reported, but he regained consciousness the next morning and was extubated and sent home a few days later. It remains to be seen whether he will resume his football career. His collapse was attributed to "commotio cordis" but this is a "diagnosis of exclusion" because no other explanation was available.45 DIC due to COVID immunization is the simplest explanation of his cardiac arrest, abnormal lung bleeding, and remarkable recovery.

Example #2
Uche Nwareri, a "die-hard vax zealot," died suddenly at age 38 in his wife's home [65]. His death was attributed to "enlarged heart with acute heart failure." This diagnosis makes no sense, because Mr. Nwareri had no history of heart failure. The simplest explanation is that abnormal DIC clot formation in small peripheral arteries sharply increased flow resistance, so that his heart became distended because it was unable to empty its contents while he was dying [48].

Example #3
Franco Harris was in good health at age 72 until he died suddenly after two COVID injections. His death was dismissively attributed to "natural causes" but COVID immunization is a better explanation [144].

Example #4
Jaylon Ferguson died suddenly at age 26 without explanation.

Example #5
Riddick Parker, Jr. collapsed and died suddenly while riding a bike at age 49.

Example #6
Alaric Jackson, age 25, had his 2022 season cut short on account of blood clots.

Example #7
Max Mitchell, age 23, also had his 2022 season cut short on account of blood clots

Example #8
Henry Anderson, age 31 had a stroke and missed several games in 2022

Example #9
Jesse Lemonier, age 25, died suddenly January 26, 2023

Treatment for COVID pneumonia and other forms of Critical Illness

Stress theory suggests the following means for managing life-threatening COVID pneumonia as well as other life-threatening critical illnesses:

1. Prevention is preferable to cure. Vitamin D minimizes the risk and severity of COVID and other viral illnesses [74, 145-149].
2. Draconian quarantine measures such as those imposed in China may dangerously exaggerate viral virulence. Such misguided crowding and confinement caused the influenza epidemic that devastated the Athenians in their war with Sparta, and the devastating "Spanish Flu" epidemic of WWI [150, 4].
3. Elective endotracheal intubation protects health care workers from the contagion, provides respiratory support as needed, and enables the measurement of inhaled gas mixtures.
4. General anesthesia with ½ MAC (minimal alveolar concentration) Isoflurane (about 1%) to prevent fear and anxiety that harmfully activates the "cognitive pathway" of the stress mechanism that generates fear and harmful sympathetic nervous activity.
5. Generous opioid supplementation of general anesthesia with modern synthetic opioids such as fentanyl, Sufentanil, or Dilaudid controls the nociception pathway, prevents spontaneous hyperventilation, and promotes beneficial hypercarbia.
6. Exhaled CO2 concentrations should be maintained in the range of 50-100 torr to preserve respiratory drive, minimize microvascular flow resistance, promote cardiac efficiency, and maximize cellular oxygenation and organ protection.
7. Avoid mechanical hyperventilation and allow spontaneous breathing whenever possible.
8. Maintain pulse oximeter readings no higher than 89 by diluting the inhaled gas mixture with nitrogen or compressed air to minimize pulmonary oxygen toxicity and maintain effective hemoglobin saturation in blood emerging from the lung. Higher readings reflect meaningless plasma saturation with oxygen.
9. Monitor the partial pressure of oxygen in peripheral tissues using transcutaneous O2/CO2 technology to confirm effective tissue oxygenation.
10. Antibiotics as needed to control secondary bacterial infestations
11. Supplement with intravenous magnesium sulphate using eclampsia protocols as needed to mitigate thrombin activity and control blood hypercoagulability.
12. Avoid transfusions with packed red blood cells to treat mild or moderate anemia. The anemia beneficially promotes blood turbulence that inhibits harmful coagulopathy and re-mobilizes red cells.
13. Monitor blood coagulability using TEG or Viscoelastograph
14. note: there is no clinically available antidote for tissue factor that can control the "tissue factor pathway" that activates the stress mechanism. The development of such a treatment would revolutionize health care [151].

Long COVID Diagnosis

The available evidence, as illustrated by Susan's case, suggests

that the risk of DIC and Long COVID increases with each additional COVID immunization [40,117]. The following tests may warn of dangerous hypercoagulability caused by COVID and its immunizations that can be treated with anticoagulants to prevent complications:

1. Increased peripheral vascular resistance caused by arterial obstruction by abnormal clots and/or capillary obstruction by micro-emboli [152].
2. Thromboelastographic (TEG) is a sensitive test of blood hypercoagulability.
3. Tissue biopsy can confirm inflammation of the vascular endothelium.
4. Angiography may detect abnormal "calamari" strands of insoluble fibrin in arteries, and heart ventricles [69].
5. Elevated d-Dimer or FSP reflects capillary gate hyperactivity [153]
6. Albuminuria reflects stress mechanism hyperactivity that disrupts the glycocalyx and releases albumen protein into the bloodstream [95-99]
7. Hyaline casts in the urine also reflect stress mechanism hyperactivity that causes soluble fibrin accumulation in glomeruli.

Long COVID Treatment

Anticoagulant therapy alleviates Long COVID symptoms [59]. Streptokinase is the ideal anticoagulant choice for Long COVID because it has been extensively tested for human safety and effectiveness. It was regarded as a "miracle drug" for treatment of myocardial infarction, pulmonary embolus, and ischemic stroke in the 1980's [154-157]. Unfortunately, it has been supplanted by coronary artery bypass surgery and angioplasty, and is no longer available in North America because it causes hypotension, which actually reflects its superior ability to disintegrate abnormal clots, open the capillary gate, and restore microvascular perfusion [158, 159]. Fortunately, streptokinase remains available from its German manufacturer [160]. Alternatives include urokinase [78,161-165]. TPA (tissue plasminogen activator), [166-169]. EDTA (Chelation therapy), Magnesium sulphate, and trisodium citrate [170-173].

Management of DIC Caused by COVID Immunization

In the event that COVID related DIC is suspected, a simple and readily available CBC test can detect DIC anemia. Blood transfusion for moderate anemia should be avoided so as to allow blood turbulence to restore arterial patency and re-mobilize red cells removed from circulation by abnormal coagulation activity. Early treatment with streptokinase or other anticoagulant medications that activate plasminogen and promote restoration of red cells to circulation should be considered.

Discussion

The stress mechanism provides an improved explanation of the pathological effects of COVID and its immunizations that illustrates the power of stress theory. It explains how the "novel" coronavirus causes viral pneumonia by attacking the pulmonary endothelium and provoking inflammation and exudates. It explains how the COVID contagion causes systemic inflammation that leads to loss of smell, taste, and hair, and why older victims and those suffering obesity and chronic illnesses suffer greater COVID severity than younger victims. It explains why the mRNA vaccinations are more dangerous than COVID itself, and why repeated vaccinations progressively exaggerate the danger. Perhaps most interesting of all, it explains a hitherto unrecognized form of DIC that causes sudden death in young, healthy victims without warning symptoms, and may provide a universal explanation of sudden death in a wide variety of circumstances such as earthquakes and other forms of severe stress and fright [174]. Lastly, it suggests improved treatments for these conditions.

Conclusion

"Medicine is a social science, and politics is nothing else but medicine on a large scale. Medicine, as a social science, as the science of human beings, has the obligation to point out problems and to attempt their theoretical solution: the politician, the practical anthropologist, must find the means for their actual solution. The physicians are the natural attorneys of the poor, and social problems fall to a large extent within their jurisdiction."
----Rudolf Virchow.

The discovery of the mammalian stress mechanism represents the triumph of 20th century medical research and the next great advance in medical and biological theory. As long anticipated, it functions as the "companion mechanism" of DNA that converts the genetic blueprint into embryological development, and then remains active for the duration of life to repair tissues and regulate organs. It enables a "unified theory of medicine" that explains physiology, pathology, and stress, and directs treatments at the cause of disease rather than its symptoms. It paves the path for productive and profitable pharmacological development that will enable a new era of human existence, free from the eternal curse of disease and premature death.

The implications of the stress mechanism exceed the bounds of medicine. It implies a unified theory of biology that resolves the disparities of Darwin, Lamarck, Baldwin and Saltation, and explains embryology, evolution, extinction, ethology, longevity, taxonomy, anatomy, sex, speciation, the Cambrian explosion, and dinosaurs.

The next great advances in medicine and biology will be the discovery of mechanisms that convert chromosomal genetic information into PAR (thrombin) receptor configurations on cell surfaces to enable embryological development, and advancing computer technology that deciphers the genome [175-180]. These discoveries will enable humans to control evolution, with implications that presently reside in the realm of science fiction. Medicine has done its job: it has provided a theoretical solution to the problem of disease that promises revolutionary advances in public health. The testing, confirmation, and implementation of this discovery now lies in the hands of power, politics, privilege, and persuasion that dominates all forms of human endeavor.

My question is: Must the blessings of stress theory await the arrival of our great-grandchildren? Why not us? Why not now?

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