

# Under What Circumstances Can an Acute myocardial infarction Be regarded as a Workrelated accident?

Multi-Causal Diseases as Work accidents

---

*Mervyn S. GotSMan, MD, FrCP, FaCC\* and Stephen Adler, JD\*\**

## Abstract

Workers compensation systems compensate for work accidents and occupational diseases. multi-causal diseases, such as heart disease, are not accidents and are not occupational diseases. they are an ordinary part of life, and heart attacks, for example, may occur without a precipitating event. however, coronary heart disease may be aggravated by unusual physical exertion or mental stress, resulting in a myocardial infarct (heart attack). if such strain or stress occurs at work, it is often compensated for by the workers compensation system. this paper asks whether the legal theories that are used to determine if a heart attack should be recognised as a work accident are consistent with modern medical knowledge on the subject. to answer this question, we must first examine the medical theory relating to the effect of an event at work on a latent illness, such as pre-existing heart disease, which must be present to produce a heart attack.

We describe events at work as ‘triggers’ of acute myocardial infarction. however, most of these triggers are not connected to the workplace. triggers occasionally occur during an unusual, sudden and acute emotional event, such as stress or unusual physical effort at work, which, therefore, are work-related. this work-related event results in ruptured plaque in a diseased coronary artery. intimal (the inner layer of the artery) disruption initiates a thromboembolic clot of the coronary artery involved. thrombus forms, obstructs the artery, coronary flow is impeded, and the distal myocardium undergoes necrosis and infarcts (an acute myocardial infarction or heart attack). thus, the sudden acute emotional event or physical effort at work has caused a heart attack, which should be compensated by the workers compensation system.

the medical and legal basis for recognising the relationship between the trigger and the heart attack are not precise. We suggest that an unusual acute stressor, or trigger, which occurs in the workplace should be the basis for recognising the infarct as a work accident. in israel, when it has been determined that a work-related trigger event contributed to the occurrence of a heart attack, the attack is recognised as a work accident. this paper describes the importance of risk factors which can cause a heart attack, the experimental, pathological, and clinical evidence of plaque rupture as a cause of heart attacks, clinical examples, and legal theories for recognising a heart attack as a work accident, with particular reference to israeli and american sources.

## A. Introduction<sup>1</sup>

Workers compensation programs compensate for work-related injuries and disabilities, while other social welfare or social insurance programs, such as disability insurance and general accident insurance, provide income for those injured and disabled by causes not related to work. since workers compensation generally provides superior benefits

---

\* former director, department of Cardiology, hadassah University medical Center, Jerusalem, israel.

\*\* Judge, President, national labour Court, Jerusalem, israel. email: [stefana@court.gov.il](mailto:stefana@court.gov.il).

<sup>1</sup> alex Chester, student, harvard University law school, assisted in preparing this article for publication.

than other social welfare or social insurance programs, there must be reasonable and clear criteria to determine when a disease with several underlying or precipitating causes is considered a work accident. also, workers compensation systems are financed by employers

and should not be burdened by compensating for non-work-related injuries. It is, therefore, essential that a workers compensation system develop tools to distinguish between work-related injuries and disabilities and those which are not work related. It is a major challenge for the legal and medical profession to determine whether a disease is caused by work and to what extent it is work-related. This determination is based upon legal and medical considerations and, therefore, this paper approaches the issue from the perspective of both professions, using heart disease (coronary atherosclerosis) and heart attacks (infarcts) as examples.

We attempt to overcome the communication difficulty between the medical and legal professions, discussing heart attacks and coronary artery disease as work accidents,<sup>1</sup> occupational diseases,<sup>2</sup> non-work-related diseases,<sup>3</sup> and multi-causal diseases.<sup>4</sup> Work accidents and occupational diseases are compensated by workers compensation programs. Non work-related diseases are not compensated. Multi-causal diseases are generally not compensated; however, sometimes, an event at work can aggravate the existing disease and, therefore, precipitate or 'trigger' the appearance or complication of an underlying disease, such as a heart attack, which is recognised as a work injury.<sup>5</sup>

The law has developed a theoretical basis to determine whether a work-related factor has triggered a heart attack. These are mainly legal presumptions, generally developed by courts or workers compensation tribunals.

We shall first explain how cardiologists understand the development of coronary artery disease and how it can develop into a heart attack. Then we will describe the legal presumptions and tests for recognition of a heart attack as a work accident. Then we will discuss if the legal assumptions are compatible with medical knowledge.

## B. MeDiCal UnDerstanDing of MyoCarDial Disease (Coronary atherosClerosis) and heart attaCks (infaRcts)

### *I. The Natural Development of Myocardial Disease*

The heart requires oxygen to survive. This oxygen is supplied by arteries which surround the heart. Coronary arterial diameter (calibre) and blood flow vary continuously to adjust for the oxygen demands of the heart muscle. Thus, the walls of these arteries are an active, muscular tube which contracts or relaxes (dilates) to maintain blood pressure. If the blood flow to the heart is blocked or reduced drastically, the heart muscle cannot function and a heart attack occurs. Such changes in the blood flow due to abnormal coronary function (pathophysiology) are determined by two factors: (1) narrowing of the artery due to excessive fatty plaque

<sup>1</sup> Work accidents arise out of and during the course of work and are usually sudden and clearly work-related. therefore, when a person is at work and his hand is injured by a machine or he or she falls from a ladder, there is no causality issue since the accident is clearly work-related.

<sup>2</sup> occupational diseases are illnesses which medical research has proven to be caused entirely or mainly by a certain type of work. for example, asbestosis is generally recognised as an occupational disease for a worker in an asbestos factory. see, eg, P Brodeur, 'annals of law, the asbestos industry on trial: 1 — a failure to Warn', *The New Yorker* (new york), 10 June 1985, 57.

<sup>3</sup> non-work-related diseases develop naturally, unrelated to events at work and, therefore, are not compensated by a workers compensation program.

<sup>4</sup> multi-causal diseases have many causes, one or more having a work connection and other causes having no work connection, such as the natural development of the disease. heart disease is a multicausal disease. these diseases are not recognised as occupational diseases but may have some work connection.

<sup>5</sup> the trigger theory has also been suggested for recognising mental accidents as work accidents in an article by stephen adler and rivka schochet. see s adler and r schochet, 'Workers' Compensation and Psychiatric injury definition' (1999) 22 *International Journal of Law and Psychiatry* 603.

(atheroma), plaque rupture and/or thrombus formation; (2) dynamic variations in coronary vascular tone due to contraction or relaxation of its muscular arterial wall.

### 1. *Narrowing of the Artery*

narrowing of an artery by the formation of fatty plaque (coronary atheroma) reduces coronary blood flow to the heart muscle (myocardium). Coronary atheroma is a process in which cholesterol or its derivatives are deposited in the wall of the artery (or other blood vessels). this may be exacerbated by fibrosis (atherosclerosis or formation of scar tissue). this process develops naturally in the normal course of people's lives. atheroma accretion starts in childhood but may be accelerated by recognised risk factors, such as high plasma ldl, low hdl, elevated triglycerides, hypertension, diabetes, cigarette smoking, homocysteinaemia or a hectic lifestyle. most people have more than one of these underlying risk factors. most risk factors are unrelated to work. this gradual process is punctuated by acute episodes (the acute anatomical incident) when the developing plaque ruptures, the continuity of the inner lining of the arterial wall is broken (endothelial damage), a clot (thrombus) forms, and blood flow to the artery is obstructed.

rupture of a plaque may be part of the natural evolution of the disease or it may be triggered by an event, such as extreme stress or an unusual physical strain. trigger factors, such as sudden stress, constrict the arterial wall, and the changing wall can hasten plaque rupture. rupture may also occur when the pressure inside the cholesterol lake increases or the cap wall is too thin or weak.<sup>6</sup> the ruptured plaque then loses its natural protective surface layer, exposing the underlying vessel wall, which causes a clot (thrombus) to form. the developing clot causes sudden and progressive obstruction of the vessel, reducing blood flow and culminating in a heart attack (total occlusion). the good news is that the body has a protective mechanism (fibrinolysis) which dissolves clots. the dynamic interaction between clot formation and dissolution determines whether the final outcome of this process will be total closing of the artery (occlusion) or partial blockage or total clot dissolution, leaving a nearly smooth artery wall or a clot which dissolves and forms again.<sup>7</sup>

2. *Dynamic Variations in Coronary Vascular Tone* the muscular cells in the coronary artery wall are sensitive to changes in oxygen tension. there is also a dynamic interaction between the secretion of local substances in the blood vessel wall and nervous influences. some substances cause muscle relaxation, while others cause contraction (dynamic narrowing). normal coronary vascular tone is when this interaction maintains an optimal diameter inside the artery (luminal) and modulates blood flow to meet the needs of the heart muscle (myocardium). abnormal coronary vascular tone occurs when the feedback mechanism (homeostatic) fails, normal function is deranged and inadequate blood flow deprives the heart muscle of an adequate oxygen supply and causes ischaemia with chest pain (angina pectoris).<sup>9</sup>

## II. *Causes of Coronary Artery Disease*

according to current medical knowledge, coronary artery disease develops during most of our lives, caused by a number of factors, the most common being genes, smoking, stress (which can be work-related or non-work-related), high cholesterol, being overweight and improper

<sup>6</sup> tissue macrophages (phagocytes) produce enzymes such as metalloproteinases or collagenases, which dissolve the interstitial collagen (fibril-forming protein constituent connective tissue) in the fibrous cap. mJ davies & aC thomas, 'Plaque fissuring — the Cause of acute myocardial infarction, sudden ischaemic death, and Crescendo angina' (1985) 53 *British Heart Journal* 363; ml gronholdt, s dalager-Pedersen and e falk, 'Coronary atherosclerosis: determinants of Plaque rupture' (1998) 19 *European Heart Journal* C24.

<sup>7</sup> ma deWood et al, 'Prevalence of total Coronary occlusion during the early hours of transmural myocardial infarction' (1980) 303 *New England Journal of Medicine* 897; ms gotsman et al, 'angiographic findings in the Coronary arteries after thrombolysis in acute myocardial infarction' (1992) 70 *American Journal of Cardiology* 715; y rozenman et al, 'early intravenous thrombolysis in acute myocardial infarction: the Jerusalem experience' (1995) 49 *International Journal of Cardiology* s21.

diet. this list is not exclusive and there may be other factors which contribute to the development of coronary heart disease. most of these factors are unrelated to work. the main factor which can be related to work is stress, which is not an accident but a gradual process occurring over years, and which has many non-work-related factors.

*III. Myocardial Infarcts May be Caused by Precipitating Events* as described above, the development of coronary artery disease is a normal part of life and can develop to an extent that a heart attack will occur naturally, without outside stimulus. Pre-existing heart disease must be present to produce a heart attack. the heart functions despite the narrowing of the artery and dynamic variations in coronary tone, which are part of normal life. however, unusual narrowing or dynamic variations can be the immediate cause of a heart attack. these unusual narrowing or variations can be influenced by triggers — specific events in one's life. these triggers change the steady state of coronary structure and blood flow and cause ischaemia or infarction. such sudden events in people with or without risk factors for coronary artery disease interact to cause arterial constriction, plaque rupture and clot formation. triggers exist in a person's normal life, unrelated to work, such as unusual physical strains (sports, sex, lifting heavy objects), stress (personal crisis, arguments, fear), circadian and weekly variations, extreme climate changes and stress related to war or other outside danger. however, there may also be triggers at work which precipitate heart attacks.<sup>10</sup>

---

9 dysfunctional endothelium is a physiological dysfunction of normal biochemical processes carried out by the endothelium. normal endothelium has a specific paracrine effect on the underlying smooth muscle cells in the arterial wall. a normal artery dilates in response to an intra-arterial injection of acetylcholine and is mediated by the production of endothelial nitric oxide (no). endothelium overlying an atheromatous plaque is dysfunctional and has a blunted response to acetylcholine, no production does not occur and the coronary artery constricts. the artery is also more sensitive to endothelin and thromboxane a<sub>2</sub> with an intense vasoconstrictive effect and does not respond to prostacycline with vasodilatation. these arteries undergo more vasoconstriction and the intense spasm is likely to cause plaque rupture. an elevated serum cholesterol also causes endothelial dysfunction and this can be reversed if treated.

10 israeli examples of myocardial infarcts triggered by work-related events, which have been recognised by the national labour Court as work accidents, include:

- i. an airline manager had arranged to fly 800 supporters of the local football team abroad for a key match. the flights were overbooked. the supporters arrived at the airport and there were insufficient available seats. this caused a violent verbal and physical altercation between the supporters and the airline manager. he felt severe chest pain, collapsed and had an acute infarct. the intense psychological reaction to the event caused plaque rupture, clot formation, obstruction of the artery and an acute myocardial infarct.
- ii. a bus driver brought his bus to the filling station on a cold morning. he noticed that he had a puncture and flat tyre. the garage worker on duty had not started his shift and refused to change the tyre. an argument erupted between the driver and the worker. the driver changed the tyre, which was unusual exercise in the cold weather. he developed severe retrosternal (behind the sternum, the breastbone) chest pain and shortly thereafter an acute myocardial infarction. the sudden unusual physical activity on a cold morning was an acute stressor.
- iii. a young television director found that his senior announcer had arrived late for a television broadcast. there was a sharp argument between the two. the announcer walked out and refused to go on the air without an apology. the director felt severe chest pain and developed an acute myocardial infarction.

how does a trigger cause the heart attack? Under conditions of extreme stress, excessive secretion of adrenaline produces profound vasoconstriction, reduction in coronary blood flow, ischaemia of cardiac muscle and damage to the muscle.<sup>8</sup> excessive vasoconstriction cracks the

---

<sup>8</sup> excessive vasoconstriction can cause profound ischaemia (restriction in blood supply) or rupture of a small soft atheromatous plaque. hypercholesterolaemia or an underlying plaque changes the function of the endothelium so that the response is abnormal. a similar effect occurs in atheromatous arteries: f Crea, JC kaski, a masori et al, 'key references on Coronary artery spasm' (1994) 89 *Circulation* 2442. adenosine diphosphate (adP) accumulates and causes vasodilatation, a classic autocrine reaction. the normal endothelium secretes several paracrine (signalling) mediators which modulate the function of the neighbouring muscle cells. increases in blood flow activate local shear stress sensitive elements in the endothelial cells. they secrete nitric oxide that diffuses to the adjacent medial muscular cells which causes relaxation with vasodilatation. the endothelium

plaque with rupture. this initiates a thrombotic cascade: thrombus forms and occludes the artery, interrupts coronary blood flow and precipitates acute myocardial infarction.<sup>9</sup> to summarise the pathophysiology of the triggering mechanisms, there

- iv. a young garage worker had to jack up a motor car and the jack slipped. the worker was under the car and he tried to prevent the car from falling by propping the car up by himself. he started to sweat. an hour later he had severe chest pain and that evening sustained an acute myocardial infarct.
- v. a young office worker was mobilised for his annual reserve army training. after three days of severe physical training with prolonged unaccustomed exercise, he developed severe retrosternal chest pain and later that evening had an acute myocardial infarction. the unusual exercise precipitated rupture of a previously insignificant plaque.
- vi. a worker in a tyre factory loaded 400 tyres onto a truck manually each day. the administration tried to improve output. on the first day of the new schedule, the worker loaded 800 tyres, and in the late afternoon he felt severe chest pain. that evening he developed an acute infarct. the sudden change in the intensity of physical work induced plaque rupture, despite the fact that the patient normally undertook heavy physical labour.
- vii. a middle-aged butcher found that two of his workers did not appear for work in the morning after repeated scud missile attacks during the Persian gulf War. he did the work of three workers during the next 12 hours, and during the day there were two further siren warnings of missile attacks. that evening he developed an acute myocardial infarct. the extra physical work and the emotional fright caused by the missile warnings induced rupture of an insignificant atheromatous (accumulation and swelling in artery walls) plaque.

in all of these cases, risk factors for atheroma accretion were present. however, in each instance, it was considered that the emotional or physical trigger was more important than the risk factor and that random chance could not explain the event. in some cases, the chest pain started during the acute emotional or physical stress. however, in some patients, it was delayed by 12 or 24 hours. Presumably the plaque ruptured during the acute stressful event, thrombus formed and was lysed and washed away, and only some hours later, the rate of thrombosis with decreased flow exceeded the rate of thrombolysis: s koubovi, *Work Accidents Issues*, israel Bar association Press, Jerusalem 1999 at 25–52.

are five underlying mechanisms involved in the trigger of a myocardial infarction: (1) intense vasoconstriction; (2) endothelial dysfunction; (3) rupture of an atheromatous plaque; (4) generation of a thrombus over the ruptured plaque, or plaque with damaged endothelium; and (5) defective thrombolysis. We suggest that the trigger event at work, which can be the immediate cause for a heart attack, is the basis for the legal principle determining when a heart attack will be recognised as a work accident.

### C. legal recognition of a heart attack, or heart Disease, as a Work Accident

most workers compensation systems recognise that coronary artery disease has many underlying causal factors and is, therefore, not an occupational disease. also, since the heart disease is not caused by an accident (one-time sudden event), it is not recognised as a work

---

also secretes prostacycline, a potent vasodilator. endothelin, also secreted by the endothelial cells, causes contraction of the arterial muscle cells with vasoconstriction and reduction in coronary blood flow.

Circulating platelets also have a paracrine effect; they secrete thromboxane a<sub>2</sub>, a potent vasoconstrictor. they also initiate and amplify the clotting mechanism in the bloodstream. renin, secreted by the juxtaglomerular (near a kidney glomerulus), converts angiotensinogen (serum globulin formed by the liver) to angiotensin i (oligopeptide in the blood that causes vasoconstriction). this is converted to angiotensin ii, a potent vasoconstrictor. this is an endocrine effect, the parent substance is secreted by the kidney but it acts on a distant effector site. adrenergic nerves liberate neurotransmitters (norepinephrine), which dilate or constrict the arteries — alpha receptors cause vasoconstriction and beta receptors vasodilatation. the parasympathetic (craniosacral) neurotransmitter, acetylcholine (the only neurotransmitter used in the somatic nervous system), causes vasodilatation (dilation of a blood vessel).

there is a dynamic interaction between these effects. some cause muscle relaxation, others contraction: the interaction maintains optimal luminal diameter of the arteries and modulates blood flow to meet the needs of the myocardium. however, when the homeostatic feedback mechanism fails, normal physiology is deranged. inadequate blood flow deprives the local muscle of an appropriate oxygen supply and causes ischaemia.

<sup>9</sup> e falk, 'Coronary thrombosis: Pathogenesis and Clinical manifestations' (1991) 68 *American Journal of Cardiology* 68. they are mediated through different mechanisms: increased sympathetic activity, endothelin secretion, thromboxane a<sub>2</sub> production and prothrombotic activity, or decreased nitric oxide production, prostacycline secretion and decreased thrombolytic activity.

accident. the fundamental principle of compensability for workers compensation is an 'injury by accident.'<sup>10</sup> an important principle of many workers compensation systems, including those in israel and most american states, accepts a worker as he is, including the risk factors which cause heart disease to develop. sudden acute stress and strain are part of a person's normal life outside of work but may also occur at the workplace. a heart attack may occur as a part of one's normal life or may be triggered by events occurring at work or outside of work. since most causes/risk factors of heart disease are not work-connected and it has not been proven that workers have a significant risk of developing heart disease more than other sectors of the population, this disease is not recognised by most jurisdictions as an occupational disease. in israel and most american states, heart disease is also not recognised as a 'micro-trauma', because it has not been proven that it is caused by many 'micro' events at work occurring over a period of time. Work stress events or recurrent physical exertion are difficult to isolate and measure as micro events. there are, however, some jurisdictions which recognise workers doing certain types of work, such as police, as having greater risks of unnatural development of heart disease.<sup>11</sup> a few jurisdictions will accept a claim regarding heart disease if a physician has testified that it was caused by the worker's work.<sup>12</sup>

When a heart attack is triggered by an unusual strain or stress at work, it should be recognised as a work accident. these are generally unexpected events. While the worker suffers from coronary artery disease, the immediate cause of the heart attack is the workrelated trigger event. such a heart attack is seen as an accident which has been triggered by an acute strain or stress at work.

When events occurring at work (a precipitating event) have a significant effect upon a heart attack, there is a justification for the workers compensation system compensating the injured worker. there is more justification for the workers compensation system compensating a worker who has had a heart attack when the work-related triggers were substantial and were a significant cause in triggering the myocardial infarct. this theory is compatible with the modern medical understanding of coronary heart disease and heart attacks, and is based upon medical research which indicates that workrelated events, especially unusual stress or strain, may trigger an acute heart attack. many jurisdictions define the trigger, or precipitating event, as an unusual strain or stress at work, which can be connected in time to the worker's heart attack. an important policy decision for a workers compensation system is to determine the type of workrelated triggers required for recognising a myocardial infarct as a work accident.

most workers compensation systems require four elements in order to recognise a myocardial infarct as a work accident: (1) a diagnosed heart attack, identifiable in time. (2) a work-related event, generally an unusual event, which is either an acute emotional or mental stress or an unusual physical strain. an 'unusual' event is required as an indication that the heart attack did not occur in the person's normal life but instead is work-connected. the main legal problem is to identify the unusual stress or strain and determine its intensity and contribution to the occurrence of the heart attack. intensity is usually determined by comparing the specific

<sup>10</sup> P Barth and ha hunt, *Workers' Compensation and Work-Related Illnesses and Diseases*, mit Press, 1980 at 105.

<sup>11</sup> see, eg, *Duffy v. Commonwealth/Dep't of State Police*, 22 Va. app. 245, 468 s.e.2d 702 (1996); *Medlin v. County of Henrico Police*, 34 Va. app. 396, 542 s.e.2d 33 (2001). in fact, even states that are most extreme in not including heart conditions under workers compensation, such as nevada, still make exceptions for such public-sector protective occupations. see P Barth and ha hunt, *Workers' Compensation and Work-Related Illnesses and Diseases*, mit Press, 1980 at 108.

<sup>12</sup> for example, illinois effectively requires a claimant only to 'find a credible medical witness to testify that the exertion might or could have been the proximate cause of the injury': P Barth and ha hunt, *Workers' Compensation and Work-Related Illnesses and Diseases*, mit Press, 1980 at 108. according to some, because the specific cause of a case of heart disease is unknown, finding a medical witness to testify is a simple matter. thus the effect of the illinois law is to remove all serious obstacles to compensation, making cases dependent only on the skill of the participating attorneys. see rV dalenberg, 'Coronary heart disease and the law' (1973) 42 *Modern Concepts of Cardiovascular Disease* 29.

event to the worker's usual daily work routine, including his usual work-related stress and strain. (3) evidence, both medical and legal, of a connection between the occurrence of the myocardial infarct, at the time it occurred, and the event at work. (4) evidence, both medical and legal, that the work-related triggers of the heart attack played more than a chance role in the occurrence at the time it happened. these slippery criteria relate to the balancing of the work-related and non-work triggers of the heart attack.

according to richard Cohen and gary klein, because medical science cannot conclusively establish causality, the test can only be a legal one. thus they propose that the test should be the difference between the employment exertion and that of a normal person, without taking into account any possible 'personal causal contribution.'<sup>13</sup>

### *I. Triggering Events at the Workplace*

there are a number of trigger events which may be the immediate cause of a heart attack and which occur during one's normal lifetime or at the workplace. these include circadian patterns, sudden physical exertion, mental stress, a return to work, sudden changes in climate and major life-threatening external influences.

#### *1. Circadian Patterns*

Circadian patterns are the normal 24-hour variations of body activity. there are major circadian variations in heart rate and blood pressure. they increase in the early morning after rising, and when one goes to work. there is a small secondary peak in the late afternoon and a major decrease at night during sleep. the body is less active at night than during the day. Changes in the circadian pattern, which can be described as heart rate and blood pressure variations and coronary artery events, can trigger a heart attack.

it has been shown that an unusually high percentage of heart attacks occur because of circadian patterns. sudden cardiac death also has a circadian variation. the framingham heart study found that sudden death was more common from nine o'clock in the morning to one o'clock in the afternoon, and the hourly risk of sudden cardiac death was 70 per cent greater between seven and nine o'clock in the morning.<sup>14</sup> in other studies adjusted for wake-up times, sudden death was greater in the first three hours after waking.<sup>15</sup> three studies have shown a primary peak incidence of infarction at nine o'clock in the morning and a secondary peak at eight o'clock in the evening.<sup>16</sup> similar changes were found in different participating countries. environmental stresses, periods of rest and changes in the temporal pattern of work also alter the circadian variation. an afternoon siesta, for example, produces a fall in heart rate and blood pressure but waking up is associated with the same changes which occur after awakening in the morning.<sup>17</sup> the circadian patterns express themselves through heart rate and blood pressure variations and coronary artery events. heart rate and blood pressure variations occur during the day and night.<sup>18</sup> the fright-flight situation stimulates the adrenergic nervous system axis and

<sup>13</sup> rs Cohen and gs klein, 'a Proposed solution to the legal Problems of Workmen's Compensation heart Cases' (1973) 1 *Supplemental Studies for the National Commission on State Workmen's Compensation Laws* 179.

<sup>14</sup> this was also shown in the Cardiac arrhythmia suppression trial (Cast): sn Willich et al, 'Circadian Variation in the incidence of sudden Cardiac death in the framingham heart study Population' (1987) 60 *American Journal of Cardiology* 801.

<sup>15</sup> sn Willich et al, 'increased onset of sudden Cardiac death in the first three hours after awakening' (1992) 70 *American Journal of Cardiology* 65. the triggers and mechanisms of myocardial infarction (trimm) study also examined shift workers and found that the peak onset of infarction occurred in the first three hours after waking.

<sup>16</sup> the multicenter investigation of limitation of infarct size (milis), the intravenous streptokinase in acute myocardial infarction (isam) and the second intravenous streptokinase and infarct survival (isis-ii).

<sup>17</sup> d mulcahy et al, 'heart rate and Blood Pressure Consequences of an afternoon siesta (snooze induced excitation of sympathetic triggered activity)' (1993) 71 *American Journal of Cardiology* 611.

<sup>18</sup> d sapoznikov, mh luria, y mahler, ms gotsman, 'day vs night eCg and heart rate Variability Patterns in Patients without obvious heart disease' (1992) 25 *Journal of Electrocardiology* 175.

causes a fast heart rate (tachycardia) and a rise in blood pressure. rest (parasympathetic stimulation), on the other hand, slows the heart rate (bradycardia). heart rate increases when one has strain or stress, even in anticipation of exercise, and falls when the body is at rest and even in anticipation of rest.<sup>19</sup> further short-term changes (minutes or hours) occur in response to daily activity or rest, or at night in response to different levels of sleep, arousal, or dreams. People with stable coronary artery disease show peak ischaemic activity in the morning hours, particularly in the first three hours after waking.<sup>20</sup>

### 2. Sudden Physical Exertion

running or isometric lifting of heavy objects causes fast heartbeat (tachycardia) and a rise in blood pressure, and may precipitate a heart attack. sudden anger and vigorous sexual activity have similar effects.<sup>21</sup> it has been shown that regular physical activity reduces the incidence of acute events precipitated by violent exercise.<sup>22</sup> two recent studies<sup>23</sup> showed that physical exertion in non-trained patients was an important precipitating factor. mountain hiking and downhill skiing also increase the incidence of sudden cardiac death for people who are not used to this physical effort.<sup>24</sup>

### 3. Mental Stress

excessive mental stress may be an important trigger of heart attacks.<sup>25</sup> mental stress is more difficult to assess objectively but the onset study showed that high anger levels were reported by 14 per cent of patients within 26 hours prior to the onset of myocardial infarction.<sup>26</sup> type A personalities (high performers) were more prone to heart attack. it has also been shown that type B blue-collar workers exhibit learned helplessness, which can trigger a heart attack.

on the london City bus lines, bus drivers have more acute myocardial infarctions than ticket takers. the drivers have a time-urgent sedentary occupation and have to adhere to strict timetables, while the conductors are non-pressurised, physically active workers.<sup>27</sup>

mental stress has also been studied in people with silent ischaemia. mental arithmetic can induce ischaemia and is associated with changes in cerebral blood flow in regions of the brain associated with mental stress.<sup>28</sup> it is also associated with an increase of a substance (local nitric oxide or no) which causes a negative response (paradoxical forearm vasodilator response) to mental stress.<sup>29</sup>

<sup>19</sup> mh luria et al, 'early heart rate Variability alterations after acute myocardial infarction' (1993) 125 *American Heart Journal* 676.

<sup>20</sup> d mulcahy et al, 'Circadian Variation of total ischaemic Burden and its alteration with anti-anginal agents' (1988) 332 *The Lancet* 755.

<sup>21</sup> Je muller et al, 'triggering myocardial infarction by sexual activity' (1996) 275 *Journal of the American Medical Association* 1405.

<sup>22</sup> sn Willich et al, 'Physical exertion as a trigger of acute myocardial infarction' (1993) 329 *New England Journal of Medicine* 1684.

<sup>23</sup> the trimm study and the onset study examined the patient's activity in the hours before acute myocardial infarction.

<sup>24</sup> m Burtcher, m Philadelphia and r likar, 'sudden Cardiac death during mounting hiking and downhill skiing' (1993) 23 *New England Journal of Medicine* 1738.

<sup>25</sup> dk ahern et al, 'Biobehavioural Variables and mortality or Cardiac arrest in the Cardiac arrhythmia Pilot study (CaPs)' (1991) 67 *American Journal of Cardiology* 121.

<sup>26</sup> ma mittleman et al, 'triggering of acute myocardial infarction onset by episodes of anger' (1995) 92 *Circulation* 1720.

<sup>27</sup> Jn morris et al, 'Coronary heart disease and Physical activity of Work' (1953) 262 *The Lancet* 1053.

<sup>28</sup> r soufer et al, 'Cerebral Cortical hyperactivation in response to mental stress in Patients with Coronary artery disease' (1998) 95 *Proceedings of the National Academy of Sciences of the USA* 6454.

<sup>29</sup> C Cardillo et al, 'role of nitric oxide in the Vasodilator response to mental stress in normal subjects' (1997) 80 *American Journal of Cardiology* 1070.

#### 4. Return to Work

there are weekly rhythms, such that more heart attacks occur when people return to work on Monday mornings and stress levels increase.

#### 5. Sudden Changes in Climate

sharp decreases or increases in temperature and changes in sunspot activity are associated with an increased prevalence of heart attacks.<sup>30</sup>

#### 6. Major Life-threatening External Influences

major life-threatening external influences precipitate heart attacks. the Athens earthquake in 1981 increased the incidence of cardiac death when compared to the same period in the previous year.<sup>31</sup> more heart attacks occurred during the 1994 California earthquake and the day after than in the subsequent week.<sup>32</sup> the threat of war has the same effect and the Scud missile attacks on Israel during the Persian Gulf War increased the incidence of cardiac death.<sup>33</sup>

### D. Discussion

most of the abovementioned triggers are not connected to work. the circadian pattern is part of our everyday activity, although sometimes work may require sudden changes in our daily routine. sexual activity, sudden climate changes, wars and earthquakes are not related to work. the triggering events which may be work-related are unusual physical strain or mental stress. these must be interpreted against the background of everyday, routine, work-related environmental stress and strain, which is present for the specific worker and also his or her predisposing, personal, underlying pathogenic mechanism.

the legal and medical knowledge appear to be compatible with each other. an accident is a sudden event which causes physical injury. if acute stress or unusual physical exertion are work-related and trigger the heart attack, it is considered a work accident. myocardial infarction is viewed as an injury related to the sudden accident, since it occurs suddenly and damages the heart. medicine accepts that an unusual strain or stress may trigger a heart attack. therefore, despite the fact that the worker suffered from coronary artery disease, which was the background for his heart attack, its immediate cause was the triggering event at work.

Unless the triggering event had an insignificant role in precipitating the heart attack, it is reasonable to place the burden of compensation for the heart attack on the source of the insurance (workers compensation system, private insurance company, et cetera). therefore, in Israel and other jurisdictions, when the work-related triggers play more than a chance role in the occurrence of the heart attack, at the time it occurred, the heart attack is regarded as work-related and compensated as a work accident.<sup>37</sup> since a significant proof that a trigger at work caused the heart attack is the time connection, for a heart attack to be recognised as a work accident in Israel, it must occur within a short time after the trigger event — minutes, hours or a few days.<sup>38</sup> this is because the plaque can rupture, which causes a clot and blockage, then repair itself and then clot again, all of which can take a few days. therefore, it is assumed that if the heart attack occurs within hours or days of the unusual strain or stress at work, there is a causal relationship between them and, therefore, the infarct should be regarded as a work accident. in Israel, the national insurance institute, which is responsible

<sup>30</sup> Ri glass and mm Zack, Jr, 'increase in deaths from ischaemic heart disease after Blizzards' (1979) 313 *The Lancet* 485.

<sup>31</sup> d trichopoulos et al, 'Psychological stress and fatal heart attack, the Athens earthquake natural experiment' (1983) 1 *The Lancet* 441.

<sup>32</sup> J leor, k Poole and ra kloner, 'sudden Cardiac death triggered by an earthquake' (1996) 334 *New England Journal of Medicine* 413.

<sup>33</sup> sr meisel et al, 'effect of iraqi missile War on incidence of acute myocardial infarction and sudden death in Israeli Civilians' (1991) 338 *The Lancet* 660.

for workers compensation payments, and the national labour Court, which decides disputes relating to labour law and social welfare law, have adopted this rule and accepted heart attack as a work-related accident which entitles the worker to compensation.<sup>39</sup> the fundamental principle is that the acute stressor should make a substantial contribution to causing the infarct and be more prominent than the natural predisposing risk factors.

Prior to the adoption of the trigger test, heart attack cases were decided according to Professor arthur larson's test, which divided workers into two categories: those with pre-disposed risks and those without predisposed risks. the worker with pre-disposed risks had to prove a more acute work event than the worker without such risks.<sup>40</sup> Because of the fact that, according to the american heart association, a heart attack is always the result of underlying conditions, there is always 'personal causal contribution'. thus, the larson test became a de facto 'wear and tear' test, as in new york.<sup>41</sup> the larson test was abandoned because of the difficulty to determine whether a worker had predisposed risks. since a heart attack occurs when coronary artery disease is present, all workers have a certain degree of predisposed risk. states then turned to an 'unusual exertion' rule, which only awarded compensation if the worker's exertion was greater than what should have been expected. But most states abandoned this stricter standard, and those that did not abandon it relaxed it, such as new york, which allowed

<sup>37</sup> *Dan Yitzhak v NIL* national labour Court, 18 lbr Ct Cases 315, 411. (hebrew).

<sup>38</sup> *Zitelli v. NIL*, national labour Court, nevo 171/2006, 19.2.2007. (hebrew).

<sup>39</sup> s koubovi, *Work Accidents Issues*, israel Bar association Press, 1999 at 25–52.

<sup>40</sup> a larson, *Workers' Compensation Law*, mathew Bender new york 1972 at 7-166.

<sup>41</sup> P Barth and ha hunt, *Workers' Compensation and Work-Related Illnesses and Diseases*, mit Press, 1980 at 112–13. see text accompanying footnote 42.

the unusual event to be measured against varying standards, including the 'wear and tear' of nonemployment activity.<sup>34</sup> this overturned the earlier rule, used in the *Dworak v. E. Greenbaum Co* case, under which an employee was not awarded compensation for a heart attack suffered after exertion deemed 'part of a day's work.'<sup>35</sup> the dworak case was overturned by *Masse v. James H. Robinson Co.*, which awarded compensation to an employee who suffered a heart attack at home a week after 'strain in the course of daily work.'<sup>36</sup>

Pennsylvania courts also distinguished between cases in which the worker had a pre-existing heart condition, in which case unusual exertion had to be shown,<sup>37</sup> though this was changed in 1972 when the legislature eliminated the 'accident' requirement.<sup>38</sup> minnesota has no 'by accident' requirement, so unusual exertion is never needed.<sup>39</sup> the law is similar in georgia.<sup>40</sup>

### *I. Lessons for the Prevention of Heart Attacks at Work*

it is possible to reduce the occurrence of heart attacks. Business and industry can, for example, modulate the environmental stressors to decrease the incidence of work-related events. While it is generally not possible to prevent sudden emotional conflicts, work which has inbuilt mental stress should be studied and modified. this includes work with learned helplessness, time urgent mental activities and stress, which have been proven to increase the level of heart attacks. also,

<sup>34</sup> *Burris v. Lewis*, 2 n.y.2d 323, 141 n.e.2d 424 (1957).

<sup>35</sup> *Dworak v. E. Greenbaum Co.*, 261 app. div. 1022, 25 n.y.s.2d 829 (1941), aff'd, 287 n.y. 555, 38 n.e.2d 224 (1941).

<sup>36</sup> *Masse v. James H. Robinson Co.*, 301 n.y. 34, 37, 92 n.e.2d 56, 57 (1950).

<sup>37</sup> see, eg, *Rovere v. Interstate Cemetery Co.*, 164 Pa. super. 233, 63 a.2d 388 (1949).

<sup>38</sup> a larson, *Workers' Compensation Law*, 1972 at 43–10.

<sup>39</sup> *Fleischer v. State of Minn. Dept. of Highways*, 247 minn. 396, 77 n.W.2d 288 (1956).

<sup>40</sup> see, eg, *Lumbermen's Mut. Cas. Co. v. Kitchens*, 81 ga. app. 470, 59 s.e.2d 270 (1950), awarding compensation to a worker who suffered heart failure after carrying less than 20 pounds; *Bussey v. Globe Indem. Co.*, 81 ga. app. 401, 59 s.e.2d 34 (1950), awarding compensation to a bookkeeper who suffered heart attack five minutes after walking up a single flight of stairs to his office.

workers should be protected from unusual physical activity, such as sudden lifting of unusual heavy weights, which can trigger a heart attack.

## *II. Cost and System Implications*

recognising heart attacks as work accidents has acute cost implications. Critics say that the loosening of american standards threatens to overburden the system, turning it into an 'all-encompassing health insurance program.'<sup>41</sup> in fact, the position of the american heart association for years was that except in rare circumstances, such as *cor pulmonale*, heart cases should be legislatively removed from the workers compensation system.<sup>42</sup> many states have adopted stricter standards out of such concerns.<sup>43</sup> social welfare programs cannot avoid balancing the justice of compensating workers for infarcts which have probably been triggered by work-related events against the considerable costs of such a policy. the result in many jurisdictions is a compromise, which recognises work-related heart attacks as work accidents, subject to certain conditions which increase the accuracy and fairness of the policy and also limit costs. a society's attitude towards enlarging the circle of accepted work injuries beyond those of work accidents and occupational diseases is also affected by its general social welfare program. in those countries with broad social welfare programs, there is less urgency to compensate for a work accident, since the worker whose heart attack is not work-related has access to disability insurance, general non-work-related accident insurance, medical coverage and more. thus, failure to recognise a heart attack as a work accident will not leave the injured worker without income. however, in jurisdictions which do not have alternate sources of income for workers disabled by a heart attack, the outcome of the compensation case may mean the difference between poverty and a minimum income which guarantees a life with human dignity. other critics have proposed a strictly medical approach, under which an impartial medical panel evaluates the merits of compensation claims.<sup>44</sup> though the panel's finding is not binding, it is conclusive in the absence of an appeal. this is similar to a plan adopted in Utah.<sup>45</sup> it also mirrors the impartial court-appointed medical advisors used in the israeli labour Court, where the medical opinion has an important role in deciding whether the heart attack was work-related.<sup>46</sup>

## *III. Conclusion Regarding Heart Attacks as a Work Accident*

We suggest that jurisdictions adopt the 'unusual-strain-and-stress trigger test' to decide whether heart attacks should be recognised as work injuries. this test is compatible with modern medical knowledge of how acute strain or stress can trigger a heart attack.

---

<sup>41</sup> ga hellmuth, 'medical studies on Workmen's Compensation' (1967) 36 *Industrial Medicine and Surgery* 604.

<sup>42</sup> see Je goshkin, 'legislative action, the only reasonable solution to the Problems of Workmen's Compensation heart Cases' (1970) 5 *Forum* 329.

<sup>43</sup> see, eg, *LaTourette v. Workers' Comp. Appeals Bd.*, 17 Cal. 4th 644 (1981): an employee who suffered a heart attack due to a pre-existing heart condition while attending a work-related seminar did not suffer a compensable injury because the injury did not arise out of the employment and had no causal connection.

<sup>44</sup> rk roberts, 'Problems and suggested solutions to Cardiovascular Cases in the Workmen's Compensation field' (1970) 6 *Williamette Law Journal*, 91.

<sup>45</sup> P Barth and ha hunt, *Workers' Compensation and Work-Related Illnesses and Diseases*, mit Press, 1980 at 114.

<sup>46</sup> see s adler, 'medical evidence in the israeli labor Courts: Court-appointed expert medical advisors' in *Hospital Law*, eds a Carmi and s schneider, springer-Verlag, new york, 1988 at 212.