DOWNLOAD PDF AN EVIDENCED-BASED APPROACH FOR ESTIMATING DECOMPRESSION SICKNESS RISK IN AIRCRAFT OPERATIONS

Chapter 1 : - NLM Catalog Result

NASA/TM An Evidenced-Based Approach for Estimating Decompression Sickness Risk in Aircraft Operations Ronald R. Robinson Joseph P. Dervay.

In extreme cases, symptoms may occur before the dive has been completed. Navy and Technical Diving International , a leading technical diver training organization, have published a table that indicates onset of first symptoms. The table does not differentiate between types of DCS, or types of symptom. It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. This term was introduced in the 19th century, when caissons under pressure were used to keep water from flooding large engineering excavations below the water table , such as bridge supports and tunnels. Workers spending time in high ambient pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure is not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. DCS is very rare in healthy individuals who experience pressures equivalent to this altitude. However, since the pressure in the cabin is not actually maintained at sea-level pressure, there is still a risk of DCS in individuals having dived recently. Also, cabin pressurization systems fail occasionally , and some people may be vulnerable to the drop in pressure that occurs even in pressurized aircraft. The higher the altitude of exposure the greater is the risk of developing altitude DCS. They may be considered as either environmental or individual. Environmental The following environmental factors have been shown to increase the risk of DCS: Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale , venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred over nitrogen in gas mixtures for deep diving. Most divers do longer decompressions; however, some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox , [54] but controlled decompression is still required to avoid DCS. Isobaric

counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. Homogeneous nucleation, where bubbles form within the liquid itself is less likely because it requires much greater pressure differences than experienced in decompression. In the body, bubbles may be located within tissues or carried along with the bloodstream. The speed of blood flow within a blood vessel and the rate of delivery of blood to capillaries perfusion are the main factors that determine whether dissolved gas is taken up by tissue bubbles or circulation bubbles for bubble growth. This will result in a shorter available time under water or an increased decompression time during the subsequent dive. The total elimination of excess gas may take many hours, and tables will indicate the time at normal pressures that is required, which may be up to 18 hours. Reduction in decompression requirements can also be gained by breathing a nitrox mix during the dive, since less nitrogen will be taken into the body than during the same dive done on air. The algorithms used are designed to reduce the probability of DCS to a very low level, but do not reduce it to zero. Breathing pure oxygen significantly reduces the nitrogen loads in body tissues by reducing the partial pressure of nitrogen in the lungs, which induces diffusion of nitrogen from the blood into the breathing gas, and this effect eventually lowers the concentration of nitrogen in the other tissues of the body. If continued for long enough, and without interruption, this provides effective protection upon exposure to low-barometric pressure environments. Therefore, it is currently used only by military flight crews and astronauts for protection during high-altitude and space operations. It is also used by flight test crews involved with certifying aircraft, and may also be used for high altitude parachute jumps. Astronauts aboard the International Space Station preparing for extra-vehicular activity EVA "camp out" at low atmospheric pressure, Neurological symptoms, pulmonary symptoms, and mottled or marbled skin lesions should be treated with hyperbaric oxygen therapy if seen within 10 to 14 days of development. At a dive site, a riskier alternative is in-water recompression. It is no longer recommended to administer aspirin, unless advised to do so by medical personnel, as analgesics may mask symptoms. People should be made comfortable and placed in the supine position horizontal , or the recovery position if vomiting occurs. However, permanent long-term injury from DCS is possible. Three-month follow-ups on diving accidents reported to DAN in showed From to , they recorded 50, dives, from which 28 recompressions were required â€” although these will almost certainly contain incidents of arterial gas embolism AGE â€” a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a viper in a vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised that this was the cause of death. Charles Pasley , who was involved in the recovery of the sunken warship HMS Royal George , commented that, of those having made frequent dives, "not a man escaped the repeated attacks of rheumatism and cold". First documented case of decompression sickness, reported by a mining engineer who observed pain and muscle cramps among coal miners working in mine shafts air-pressurized to keep water out. Bauer published outcomes of 25 paralyzed caisson workers. From to , all prominent features were established. Explanations at the time included: The Eads Bridge in St Louis employed compressed air workers including Alphonse Jaminet as the physician in charge. There were 30 seriously injured and 12 fatalities. Jaminet developed decompression sickness and his personal description was the first such recorded. The similarity between decompression sickness and iatrogenic air embolism as well as the relationship between inadequate decompression and decompression sickness was noted by Friedburg. Andrew Smith first utilized the term "caisson disease" describing cases of decompression sickness as the physician in charge during construction of the Brooklyn Bridge. Recompression treatment was not used. The project chief engineer Washington Roebling suffered from caisson disease. He battled the after-effects of

the disease for the rest of his life. During this project, decompression sickness became known as "The Grecian Bends" or simply "the bends" because afflicted individuals characteristically bent forward at the hips: Leonard Hill used a frog model to prove that decompression causes bubbles and that recompression resolves them. Tunnel building to and from Manhattan caused over 3, injuries and over 30 deaths which lead to laws requiring PSI limits and decompression rules for "sandhogs" in the United States [95] Seibe and Gorman in conjunction with Leonard Hill develop and produce a closed bell in which a diver can be decompressed at the surface. Experimental decompression chambers were in use on land and aboard ship. The US Navy published the first standardized recompression procedure. Behnke introduced the "no-stop" decompression tables. Altitude DCS is treated with hyperbaric oxygen for the first time. Robert Workman established a new method for calculation of decompression requirements M-values. The "SOS Decompression Meter", a submersible mechanical device that simulated nitrogen uptake and release, was introduced. FC Golding et al. Orca produced the "EDGE", a personal dive computer, using a microprocessor to calculate nitrogen absorption for twelve tissue compartments. This is because scuba diving is considered an elective and "high-risk" activity and treatment for decompression sickness is expensive. A typical stay in a recompression chamber will easily cost several thousand dollars, even before emergency transportation is included. Although nitrogen diffuses more slowly than helium, nitrogen is much more soluble than helium and the total inert gas load in some tissues can temporarily exceed the critical supersaturation limit, resulting in bubble formation. The inner ear is particularly susceptible to this effect.

Chapter 2 : Decompression sickness : Wikis (The Full Wiki)

Estimating the risk of decompression Sickness (DCS) in aircraft operations remains a challenge, making the reduction of this risk through the development of operationally acceptable.

DCS is caused by a reduction in ambient pressure that results in the formation of bubbles of inert gases within tissues of the body. It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. This term was introduced in the 19th century, when caissons under pressure were used to keep water from flooding large engineering excavations below the water table , such as bridge supports and tunnels. Workers spending time in high ambient pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure is not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. DCS is very rare in healthy individuals who experience pressures equivalent to this altitude. However, since the pressure in the cabin is not actually maintained at sea-level pressure, there is still a risk of DCS in individuals having dived recently. Also, cabin pressurization systems fail occasionally , and some people may be vulnerable to the drop in pressure that occurs even in pressurized aircraft. The higher the altitude of exposure the greater is the risk of developing altitude DCS. They may be considered as either environmental or individual. Decompression sickness and arterial gas embolism in recreational diving are associated with certain demographic, environmental, and dive style factors. A statistical study published in tested potential risk factors: No significant associations with risk of decompression sickness or arterial gas embolism were found for asthma, diabetes, cardiovascular disease, smoking, or body mass index. Increased depth, previous DCI, larger number of consecutive days diving, and being male were associated with higher risk for decompression sickness and arterial gas embolism. Nitrox and drysuit use, greater frequency of diving in the past year, increasing age, and years since certification were associated with lower risk, possibly as indicators of more extensive training and experience. Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale , venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium

both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred over nitrogen in gas mixtures for deep diving. Most divers do longer decompressions; however, some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox , [56] but controlled decompression is still required to avoid DCS. Isobaric counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. Homogeneous nucleation, where bubbles form within the liquid itself is less likely because it requires much greater pressure differences than experienced in decompression. In the body, bubbles may be located within tissues or carried along with the bloodstream. The speed of blood flow within a blood vessel and the rate of delivery of blood to capillaries perfusion are the main factors that determine whether dissolved gas is taken up by tissue bubbles or circulation bubbles for bubble growth. This will result in a shorter available time under water or an increased decompression time during the subsequent dive. 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A US Navy treatment table 5 can be safely performed without air breaks if a built-in breathing system is not available. However, permanent long-term injury from DCS is possible. Three-month follow-ups on diving accidents reported to DAN in showed From to , they recorded 50, dives, from which 28 recompressions were required â€” although these will almost certainly contain incidents of arterial gas embolism AGE â€” a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a viper in a vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised that this was the cause of death. Charles Pasley , who was involved in the recovery of the sunken warship HMS Royal George , commented that, of those having made frequent dives, "not a man escaped the repeated attacks of rheumatism and cold". 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Chapter 3 : Uncontrolled decompression - The Full Wiki

July NASA/TMâ€" An Evidenced-Based Approach for Estimating Decompression Sickness Risk in Aircraft Operations Ronald R. Robinson.

For example, an aircraft cabin at high altitude, a spacecraft , or a hyperbaric chamber. For the catastrophic failure of other pressure vessels used to contain gas , liquids , or reactants under pressure, the term explosion is more commonly used, or other specialised terms such as BLEVE may apply to particular situations. Decompression can occur due to structural failure of the pressure vessel, or failure of the compression system itself. The Federal Aviation Administration recognizes three distinct types of decompression events in aircraft: After an explosive decompression within an aircraft, a heavy fog may immediately fill the interior as the relative humidity of cabin air rapidly changes as the air cools and condenses. Military pilots with oxygen masks have to pressure-breathe, whereby the lungs fill with air when relaxed, and effort has to be exerted to expel the air again. Slow decompression Slow, or gradual, decompression occurs slowly enough to go unnoticed and might only be detected by instruments. An example of this is the Helios Airways Flight crash, in which the pilots failed to check the aircraft was pressurising automatically and then react to the warnings that the aircraft was depressurising. Pressure vessel seals and testing Seals in high-pressure vessels are also susceptible to explosive decompression; the O-rings or rubber gaskets used to seal pressurised pipelines tend to become saturated with high-pressure gases. If the pressure inside the vessel is suddenly released, then the gases within the rubber gasket may expand violently, causing blistering or explosion of the material. For this reason, it is common for military and industrial equipment to be subjected to an explosive decompression test before it is certified as safe for use. Fallacies Exposure to a vacuum causes the body to explode This persistent myth is based on a failure to distinguish between two types of decompression: The first type, a sudden change from normal atmospheric pressure to a vacuum, is the more common. Research and experience in space exploration and high-altitude aviation have shown that while exposure to vacuum causes swelling, human skin is tough enough to withstand the drop of one atmosphere although the resulting hypoxia will cause unconsciousness after a few seconds. The second type is rare, since the only normal situation in which it can occur is during decompression after deep-sea diving. In fact, there is only a single well-documented occurrence: Neither of these incidents would have been possible if the pressure drop had been only from normal atmosphere to a vacuum. Bullets cause explosive decompression Aircraft fuselages are designed with ribs to prevent tearing; the size of the hole is one of the factors that determines the speed of decompression, and a bullet hole is too small to cause rapid or explosive decompression. A small hole will blow people out of a fuselage The television program Mythbusters examined this belief informally using a pressurised aircraft and several scale tests. The Mythbusters approximations suggested that fuselage design does not allow this to happen. Hypoxia is the most serious risk associated with decompression, especially as it may go undetected or incapacitate the aircrew.

Chapter 4 : Decompression sickness

An evidenced based approach for estimating decompression sickness risk in aircraft operations.

DCS is caused by a reduction in ambient pressure that results in the formation of bubbles of inert gases within tissues of the body. It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth[edit] DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. This term was introduced in the 19th century, when caissons under pressure were used to keep water from flooding large engineering excavations below the water table , such as bridge supports and tunnels. Workers spending time in high ambient pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure is not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. AMS results not from the formation of bubbles from dissolved gasses in the body but from exposure to a low partial pressure of oxygen and alkalosis. However, passengers in unpressurized aircraft at high altitude may also be at some risk of DCS. Symptoms of DCS in healthy individuals are subsequently very rare unless there is a loss of pressurization or the individual has been diving recently. They may be considered as either environmental or individual. Decompression sickness and arterial gas embolism in recreational diving are associated with certain demographic, environmental, and dive style factors. A statistical study published in tested potential risk factors: No significant associations with risk of decompression sickness or arterial gas embolism were found for asthma, diabetes, cardiovascular disease, smoking, or body mass index. Increased depth, previous DCI, larger number of consecutive days diving, and being male were associated with higher risk for decompression sickness and arterial gas embolism. Nitrox and drysuit use, greater frequency of diving in the past year, increasing age, and years since certification were associated with lower risk, possibly as indicators of more extensive training and experience. Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Physiology of decompression Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale , venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred

over nitrogen in gas mixtures for deep diving. Most divers do longer decompressions; however, some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox , [56] but controlled decompression is still required to avoid DCS. Isobaric counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. Homogeneous nucleation, where bubbles form within the liquid itself is less likely because it requires much greater pressure differences than experienced in decompression. In the body, bubbles may be located within tissues or carried along with the bloodstream. The speed of blood flow within a blood vessel and the rate of delivery of blood to capillaries perfusion are the main factors that determine whether dissolved gas is taken up by tissue bubbles or circulation bubbles for bubble growth. Various hypotheses have been put forward for the nucleation and growth of bubbles in tissues, and for the level of supersaturation which will support bubble growth. The earliest bubble formation detected is subclinical intravascular bubbles detectable by doppler ultrasound in the venous systemic circulation. The presence of these "silent" bubbles is no guarantee that they will persist and grow to be symptomatic. If this is severe, the symptom called "chokes" may occur. If these bubbles are not absorbed in the arterial plasma and lodge in systemic capillaries they will block the flow of oxygenated blood to the tissues supplied by those capillaries, and those tissues will be starved of oxygen. Moon and Kisslo concluded that "the evidence suggests that the risk of serious neurological DCI or early onset DCI is increased in divers with a resting right-to-left shunt through a PFO. There is, at present, no evidence that PFO is related to mild or late onset bends. In this case, the bubbles can distort and permanently damage the tissue. Direct expansion causes tissue damage, with the release of histamines and their associated affects. Biochemical damage may be as important as, or more important than mechanical effects. Platelets accumulate in the vicinity of bubbles. Endothelial damage may be a mechanical effect of bubble pressure on the vessel walls, a toxic effect of stabilised platelet aggregates and possibly toxic effects due to the association of lipids with the air bubbles. Gas is dissolved in all tissues, but decompression sickness is only clinically recognised in the central nervous system, bone, ears, teeth, skin and lungs. A catastrophic pressure reduction from saturation produces explosive mechanical disruption of cells by local effervescence, while a more gradual pressure loss tends to produce discrete bubbles accumulated in the white matter, surrounded by a protein layer. Infarcts are characterised by a region of oedema , haemorrhage and early myelin degeneration, and are typically centred on small blood vessels. The lesions are generally discrete. Oedema usually extends to the adjacent grey matter. Microthrombi are found in the blood vessels associated with the infarcts. The lipid phagocytes are later replaced by a cellular reaction of astrocytes. Vessels in surrounding areas remain patent but are collagenised. There is still uncertainty regarding the aetiology of decompression sickness damage to the spinal cord. The initial damage is attributed to the formation of bubbles, and one episode can be sufficient, however incidence is sporadic and generally associated with relatively long periods of hyperbaric exposure and aetiology is uncertain. Early identification of lesions by radiography is not possible, but over time areas of radiographic opacity develop in association with the damaged bone.

Chapter 5 : Decompression sickness - Infogalactic: the planetary knowledge core

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Uncontrolled decompression Uncontrolled decompression is an unplanned drop in the pressure of a sealed system, such as an aircraft cabin , and typically results from human error , material fatigue , engineering failure, or impact , causing a pressure vessel to vent into its lower-pressure surroundings or fail to pressurize at all. Such decompression may be classed as Explosive, Rapid, or Slow: Explosive decompression ED is violent, the decompression being too fast for air to safely escape from the lungs. Rapid decompression, while still fast, is slow enough to allow the lungs to vent. Slow or gradual decompression occurs so slowly that it may not be sensed before hypoxia sets in. Description The term uncontrolled decompression here refers to the unplanned depressurisation of vessels that are occupied by people; for example, a pressurised aircraft cabin at high altitude, a spacecraft , or a hyperbaric chamber. For the catastrophic failure of other pressure vessels used to contain gas , liquids , or reactants under pressure, the term explosion is more commonly used, or other specialised terms such as BLEVE may apply to particular situations. Decompression can occur due to structural failure of the pressure vessel, or failure of the compression system itself. The US Federal Aviation Administration recognizes three distinct types of decompression events in aircraft: After an explosive decompression within an aircraft, a heavy fog may immediately fill the interior as the relative humidity of cabin air rapidly changes as the air cools and condenses. Military pilots with oxygen masks have to pressure-breathe, whereby the lungs fill with air when relaxed, and effort has to be exerted to expel the air again. Gradual decompression Slow, or gradual, decompression occurs slowly enough to go unnoticed and might only be detected by instruments. An example of this is the Helios Airways Flight crash, in which the pilots failed to check the aircraft was pressurising automatically and then to react to the warnings that the aircraft was depressurising, eventually losing consciousness along with most of the passengers and crew from hypoxia. Pressure vessel seals and testing Seals in high-pressure vessels are also susceptible to explosive decompression; the O-rings or rubber gaskets used to seal pressurised pipelines tend to become saturated with high-pressure gases. If the pressure inside the vessel is suddenly released, then the gases within the rubber gasket may expand violently, causing blistering or explosion of the material. For this reason, it is common for military and industrial equipment to be subjected to an explosive decompression test before it is certified as safe for use. Myths Exposure to a vacuum causes the body to explode This persistent myth is based on a failure to distinguish between two types of decompression: The first type, a sudden change from normal atmospheric pressure to a vacuum, is the more common. Research and experience in space exploration and high-altitude aviation have shown that while exposure to vacuum causes swelling, human skin is tough enough to withstand the drop of one atmosphere , although the resulting hypoxia will cause unconsciousness after a few seconds. The second type is rare, since the only normal situation in which it can occur is during decompression after deep-sea diving. In fact, there is only a single well-documented occurrence: Neither of these incidents would have been possible if the pressure drop had been only from normal atmosphere to a vacuum. Bullets cause explosive decompression Aircraft fuselages are designed with ribs to prevent tearing; the size of the hole is one of the factors that determine the speed of decompression, and a bullet hole is too small to cause rapid or explosive decompression. A small hole will blow people out of a fuselage The television program Mythbusters examined this belief informally using a pressurised aircraft and several scale tests. The Mythbusters approximations suggested that fuselage design does not allow this to happen. Decompression injuries NASA candidate Astronauts being monitored for signs of hypoxia during training in an altitude chamber. The following physical injuries may be associated with decompression incidents: Hypoxia is the most serious risk associated with decompression, especially as it may go undetected or incapacitate the aircrew.

Chapter 6 : Decompression sickness | Revolvy

Abstract. Estimating the risk of decompression Sickness (DCS) in aircraft operations remains a challenge, making the reduction of this risk through the development of operationally acceptable denitrogenation schedules difficult.

In extreme cases, symptoms may occur before the dive has been completed. Navy and Technical Diving International , a leading technical diver training organization, have published a table that indicates onset of first symptoms. The table does not differentiate between types of DCS, or types of symptom. It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. Workers spending time in high-pressure atmospheric pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure surrounding them was not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. DCS is very rare in healthy individuals who experience pressures equivalent to this altitude. However, since the pressure in the cabin is not actually maintained at sea-level pressure, there is still a risk of DCS in individuals having dived recently. Also, cabin pressurization systems still fail occasionally , and some people may be predisposed to the drop in pressure that occurs even in pressurized aircraft. The higher the altitude of exposure the greater is the risk of developing altitude DCS. They may be considered as either environmental or individual. Environmental The following environmental factors have been shown to increase the risk of DCS: Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale , venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred over nitrogen in gas mixtures for deep diving. Most divers do longer decompressions, however some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox , [54] but controlled decompression is still required to avoid DCS. Isobaric counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is

known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. This will result in a shorter available time under water or an increased decompression time during the subsequent dive. The total elimination of excess gas may take many hours, and tables will indicate the time at normal pressures that is required, which may be up to 18 hours. Reduction in decompression requirements can also be gained by breathing a nitrox mix during the dive, since less nitrogen will be taken into the body than during the same dive done on air. The algorithms used are designed to reduce the probability of DCS to a very low level, but do not reduce it to zero. Breathing pure oxygen significantly reduces the nitrogen loads in body tissues and, if continued without interruption, provides effective protection upon exposure to low-barometric pressure environments. Therefore, it is currently used only by military flight crews and astronauts for protection during high-altitude and space operations. It is also used by flight test crews involved with certifying aircraft. Astronauts aboard the International Space Station preparing for extra-vehicular activity EVA "camp out" at low atmospheric pressure, Neurological symptoms, pulmonary symptoms, and mottled or marbled skin lesions should be treated with hyperbaric oxygen therapy if seen within 10 to 14 days of development. At a dive site, a riskier alternative is in-water recompression. It is no longer recommended to administer aspirin, unless advised to do so by medical personnel, as analgesics may mask symptoms. People should be made comfortable and placed in the supine position horizontal , or the recovery position if vomiting occurs. However, permanent long-term injury from DCS is possible. Three-month follow-ups on diving accidents reported to DAN in showed From to , they recorded 50, dives, from which 28 recompressions were required â€” although these will almost certainly contain incidents of arterial gas embolism AGE â€” a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a viper in a vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised that this was the cause of death. Charles Pasley , who was involved in the recovery of the sunken warship HMS Royal George , commented that, of those having made frequent dives, "not a man escaped the repeated attacks of rheumatism and cold". First documented case of decompression sickness, reported by a mining engineer who observed pain and muscle cramps among coal miners working in mine shafts air-pressurized to keep water out. Bauer published outcomes of 25 paralyzed caisson workers. From to , all prominent features were established. Explanations at the time included: Alphonse Jaminet as the physician in charge. There were 30 seriously injured and 12 fatalities. Jaminet developed decompression sickness and his personal description was the first such recorded. The similarity between decompression sickness and iatrogenic air embolism as well as the relationship between inadequate decompression and decompression sickness was noted by Friedburg. He suggested that intravascular gas was released by rapid decompression and recommended: Andrew Smith first utilized the term "caisson disease" describing cases of decompression sickness as the physician in charge during construction of the Brooklyn Bridge. Recompression treatment was not used. The project chief engineer Washington Roebling suffered from caisson disease. He battled the after-effects of the disease for the rest of his life. During this project, decompression sickness became known as "The [Grecian] Bends" because afflicted individuals characteristically arched their backs: Leonard Hill used a frog model to prove that decompression causes bubbles and that recompression resolves them. The decompression chamber was invented by the Italian engineer Alberto Gianni. The US Navy published the first standardized recompression procedure. Altitude DCS is treated with hyperbaric oxygen for the first time. Robert Workman established a new method for calculation of decompression requirements M-values. The "SOS Decompression Meter", a submersible mechanical device that simulated nitrogen uptake and release, was introduced. FC Golding et al. Orca produced the "EDGE", a personal dive computer, using a microprocessor to calculate nitrogen absorption for twelve tissue compartments. This is because scuba diving is considered an elective and

"high-risk" activity and treatment for decompression sickness is expensive. A typical stay in a recompression chamber will easily cost several thousand dollars, even before emergency transportation is included.

Chapter 7 : Decompression sickness - Wikipedia

Title(s): An evidence-based [correction of evidenced-based] approach for estimating decompression sickness risk in aircraft operations/ R.R. Robinson, J.P. Dervay, J.

Workers who spend time in high-pressure atmospheric pressure conditions are at risk when they return to the lower pressure outside the caisson without slowly reducing the surrounding pressure. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. Some divers seem more susceptible than others under identical conditions. There have been known cases of bends in snorkellers who have made many deep dives in succession. DCS may be the cause of the disease taravana which affects South Pacific island natives who for centuries have dived by breath-holding for food and pearls. The rate and duration of gas absorption under pressure. The rate and duration of outgassing on depressurization. The faster the ascent and the shorter the interval between dives the less time there is for absorbed gas to be offloaded safely through the lungs, causing these gases to precipitate come out of solution and form "micro bubbles" in the blood. Large bubbles of gas impede the flow of oxygen-rich blood to the brain , central nervous system and other vital organs. Even when the change in pressure causes no immediate symptoms, rapid pressure change can cause permanent bone injury called dysbaric osteonecrosis DON "bone cell death from pressure changes ". DON often affects the humerus and femoral heads and can be diagnosed from lesions visible in X-ray images of the bones. Cabin pressurization now prevents most DCS at altitude but cabin presurization systems still fail occasionally and some people may be predisposed to the minor drop in pressure that still occurs even in pressurized aircraft. DCS is very rare in healthy individuals who experience pressures equivalent to this altitude or less. However, since the pressure in the cabin is not actually maintained at sea-level pressure, there is still a small risk of DCS in susceptible individuals such as recent divers see Scuba diving before flying below. Predisposing factors Environmental Magnitude of the pressure reduction ratio: A large pressure reduction ratio is more likely to cause DCS than a small one. Repetitive dives within a short period of time a few hours increase the risk of developing DCS. The faster the ascent, the greater the risk of developing DCS. The longer the duration of the dive, the greater is the risk of DCS. Divers who ascend to altitude soon after a dive increase their risk of developing DCS even if the dive itself was within the dive table safe limits. Dive tables make very specific provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore the dive table surface interval is not followed if flying before that surface interval and an otherwise safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation Age: There are some reports indicating a higher risk of altitude DCS with increasing age. There is some indication that recent joint or limb injuries may predispose individuals to developing decompression related bubbles. There is some evidence suggesting that individual exposure to very cold ambient temperatures may increase the risk of altitude DCS. Typically, a person who has a high body fat content is at greater risk of DCS. While conventional wisdom would have one believe that the after-effects of alcohol consumption increase the susceptibility to DCS through increased dehydration [26] , one study concluded that alcohol consumption did not increase the risk of DCS. A hole between the atrial chambers of the heart in the fetus is normally closed by a flap with the first breaths at birth. In up to 20 percent of adults the flap does not seal, however, allowing blood through the hole when coughing or other activities raise chest pressure. In diving, this can allow venous blood with microbubbles of inert gas to return directly to the arteries including arteries to the brain, spinal cord and heart rather than pass through the lungs, where the bubbles would otherwise be filtered out by the lung capillary system [36]. In the arterial system, bubbles arterial gas embolism are far more dangerous because they block circulation and cause infarction tissue death, due to local loss of blood flow. In the brain, infarction results in stroke , in the spinal cord it may result in paralysis , and in the heart it results in myocardial infarction heart attack. Mechanism Depressurisation of the

body causes excess inert gases , which were dissolved in body liquids and tissues while the body was under higher pressure , to come out of physical solution as the pressure reduces and form gas bubbles within the body. The main inert gas for those who breathe air is nitrogen. The bubbles result in the symptoms of decompression sickness. Some of the gas goes into solution with the liquid due to the higher pressure. When the container is opened, the free gas can be heard escaping from the container and bubbles form in the liquid. These bubbles are the previously dissolved carbon dioxide gas coming out of solution as a result of the reduction to atmospheric pressure of the gas inside the container. Similarly, inert gases are dissolved in body tissues and liquids while the body is under pressure, say during a scuba dive at depth. On ascent from the dive, the excess inert gas comes out of solution in a process called " outgassing " or "offgassing". Normally most offgassing occurs by gas exchange at the lungs during exhalation [9] [38] [39]. If inert gas is forced to come out of solution too quickly to allow outgassing at the lungs then bubbles may form in the blood stream or within solid tissues inside the body. This causes the signs and symptoms of DCS which includes itching skin, rashes , joint pain and neurological disturbance. The formation of bubbles in the skin or joints results in the milder symptoms, while large numbers of bubbles in the venous blood can cause pulmonary lung damage. The most severe types of DCS interruptâ€"and ultimately damageâ€"spinal cord nerve function, which may lead to paralysis , sensory system failure, and death. In the presence of a right-to-left shunt, such as a patent foramen ovale PFO , venous bubbles may migrate to the arterial system, resulting in an arterial gas embolism which may damage the brain. Nitrogen is not the only breathing gas that causes DCS. Gas mixtures such as trimix and heliox include helium , which can also be implicated in decompression sickness. Helium both enters and leaves the body faster than nitrogen, and for dives of three or more hours in duration, the body almost reaches saturation of helium. For such dives the decompression time is shorter than for nitrogen-based breathing gases such as air. Most divers do longer decompressions, whereas some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox , [41] but controlled decompression is still required to avoid DCS. Isobaric counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is known as isobaric counterdiffusion. A gas embolism caused by the mechanical introduction of gas into the bloodstream such as via a pulmonary barotrauma injury can have many of the same symptoms as DCS. Avoiding decompression sickness is not an exact science. Accidents can occur after relatively shallow and short dives. To reduce the risks, divers should avoid long and deep dives and should ascend slowly. Also, dives requiring decompression stops and dives with less than a 16 hour interval since the previous dive increase the risk of DCS. There are many additional risk factors, such as age, obesity, fatigue, use of alcohol, dehydration and a patent foramen ovale. In addition, flying at high altitude less than 24 hours after a dive can be a precipitating factor for decompression illness. Decompression time can be significantly shortened by breathing rich nitrox or pure oxygen in very shallow water during the decompression phase of the dive. Reduction in decompression requirements can be gained by breathing a nitrox mix during the dive, since less nitrogen will be taken into the body than during the same dive done on air. Effects of breathing pure oxygen Breathing pure oxygen to remove nitrogen from the bloodstream One of the most significant breakthroughs in altitude DCS research was oxygen pre-breathing. Breathing pure oxygen before exposure to a low-barometric pressure environment decreases the risk of developing altitude DCS. Oxygen pre-breathing reduces the nitrogen loading in body tissues. Pre-breathing pure oxygen before starting ascent to altitude reduces the risk of altitude DCS. However, oxygen pre-breathing has to be continued without interruption with in-flight, pure oxygen to provide effective protection against altitude DCS. Although pure oxygen pre-breathing is an effective method to protect against altitude DCS, it is logistically complicated and expensive for the protection of civil aviation flyers, either commercial or private. Therefore, it is only used now by military flight crews and astronauts for their protection during high altitude and space operations. It is also used by flight test crews involved with certifying aircraft. Their spacesuits can operate at 4. Neurological DCS, the "chokes," and skin bends with mottled or marbled skin lesions see Table 1 should be treated with

hyperbaric oxygen therapy if seen with 10 to 14 days of development. In diving, a more risky alternative is in-water recompression. However, permanent long term injury from DCS is possible. Three month follow-up from diving accidents reported to DAN in showed From to they recorded 50, dives, from which 28 recompressions were requiredâ€”although these will almost certainly contain incidents of arterial gas embolism AGE â€”a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a viper in a vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised this was the cause of death. Colonel William Pasley who was involved in the recovery of the sunk warship HMS Royal George commented that of those who had made frequent dives "not a man escaped the repeated attacks of rheumatism and cold". First documented case of decompression sickness, reported by a mining engineer who observed pain and muscle cramps among coal miners working in mine shafts air-pressurized to keep water out. Bauer published outcomes of 25 paralyzed caisson workers. From to all prominent features were established. Explanations at the time included: Alphonse Jaminet as the physician in charge. There were 30 seriously injured and 12 fatalities. Jaminet developed decompression sickness and his personal description was the first such recorded. The similarity between decompression sickness and iatrogenic air embolism as well as the relationship between inadequate decompression and decompression sickness was noted by Friedburg. He suggested that intravascular gas was released by rapid decompression and recommended: Andrew Smith first utilized the term "caisson disease" describing cases of decompression sickness as the physician in charge during construction of the Brooklyn Bridge. Recompression treatment was not used. The project chief engineer Washington Roebling suffered from caisson disease. He battled the after-effects of the disease for the rest of his life. During this project, decompression sickness became known as "The [Grecian] Bends" because afflicted individuals characteristically arched their backs: Leonard Hill used a frog model to prove that decompression causes bubbles and that recompression resolves them. Haldane , Boycott and Damant recommending staged decompression. The US Navy published the first standardized recompression procedure.

Chapter 8 : Uncontrolled decompression - Wikipedia

Abstract: Estimating the risk of decompression Sickness (DCS) in aircraft operations remains a challenge, making the reduction of this risk through the development of operationally acceptable denitrogenation schedules difficult.

It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. This term was introduced in the 19th century, when caissons under pressure were used to keep water from flooding large engineering excavations below the water table , such as bridge supports and tunnels. Workers spending time in high ambient pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure is not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. AMS results not from the formation of bubbles from dissolved gasses in the body but from exposure to a low partial pressure of oxygen and alkalosis. However, passengers in unpressurized aircraft at high altitude may also be at some risk of DCS. Symptoms of DCS in healthy individuals are subsequently very rare unless there is a loss of pressurization or the individual has been diving recently. They may be considered as either environmental or individual. Decompression sickness and arterial gas embolism in recreational diving are associated with certain demographic, environmental, and dive style factors. A statistical study published in tested potential risk factors: No significant associations with risk of decompression sickness or arterial gas embolism were found for asthma, diabetes, cardiovascular disease, smoking, or body mass index. Increased depth, previous DCI, larger number of consecutive days diving, and being male were associated with higher risk for decompression sickness and arterial gas embolism. Nitrox and drysuit use, greater frequency of diving in the past year, increasing age, and years since certification were associated with lower risk, possibly as indicators of more extensive training and experience. Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale , venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred over nitrogen in gas mixtures for deep diving. Most divers do longer

decompressions; however, some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen -oxygen mixtures hydrox ,[56] but controlled decompression is still required to avoid DCS. This is known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. Homogeneous nucleation, where bubbles form within the liquid itself is less likely because it requires much greater pressure differences than experienced in decompression. In the body, bubbles may be located within tissues or carried along with the bloodstream. The speed of blood flow within a blood vessel and the rate of delivery of blood to capillaries perfusion are the main factors that determine whether dissolved gas is taken up by tissue bubbles or circulation bubbles for bubble growth. Various hypotheses have been put forward for the nucleation and growth of bubbles in tissues, and for the level of supersaturation which will support bubble growth. The earliest bubble formation detected is subclinical intravascular bubbles detectable by doppler ultrasound in the venous systemic circulation. The presence of these "silent" bubbles is no guarantee that they will persist and grow to be symptomatic. If this is severe, the symptom called "chokes" may occur. If these bubbles are not absorbed in the arterial plasma and lodge in systemic capillaries they will block the flow of oxygenated blood to the tissues supplied by those capillaries, and those tissues will be starved of oxygen. Moon and Kisslo concluded that "the evidence suggests that the risk of serious neurological DCI or early onset DCI is increased in divers with a resting right-to-left shunt through a PFO. There is, at present, no evidence that PFO is related to mild or late onset bends. In this case, the bubbles can distort and permanently damage the tissue. Direct expansion causes tissue damage, with the release of histamines and their associated affects. Biochemical damage may be as important as, or more important than mechanical effects. Platelets accumulate in the vicinity of bubbles. Endothelial damage may be a mechanical effect of bubble pressure on the vessel walls, a toxic effect of stabilised platelet aggregates and possibly toxic effects due to the association of lipids with the air bubbles. Gas is dissolved in all tissues, but decompression sickness is only clinically recognised in the central nervous system, bone, ears, teeth, skin and lungs. A catastrophic pressure reduction from saturation produces explosive mechanical disruption of cells by local effervescence, while a more gradual pressure loss tends to produce discrete bubbles accumulated in the white matter, surrounded by a protein layer. Infarcts are characterised by a region of oedema , haemorrhage and early myelin degeneration, and are typically centred on small blood vessels. The lesions are generally discrete. Oedema usually extends to the adjacent grey matter. Microthrombi are found in the blood vessels associated with the infarcts. The lipid phagocytes are later replaced by a cellular reaction of astrocytes. Vessels in surrounding areas remain patent but are collagenised. There is still uncertainty regarding the aetiology of decompression sickness damage to the spinal cord. The initial damage is attributed to the formation of bubbles, and one episode can be sufficient, however incidence is sporadic and generally associated with relatively long periods of hyperbaric exposure and aetiology is uncertain. Early identification of lesions by radiography is not possible, but over time areas of radiographic opacity develop in association with the damaged bone. This will result in a shorter available time under water or an increased decompression time during the subsequent dive. The total elimination of excess gas may take many hours, and tables will indicate the time at normal pressures that is required, which may be up to 18 hours. Reduction in decompression requirements can also be gained by breathing a nitrox mix during the dive, since less nitrogen will be taken into the body than during the same dive done on air. The algorithms used are designed to reduce the probability of DCS to a very low level, but do not reduce it to zero. Breathing pure oxygen significantly reduces the nitrogen loads in body tissues by reducing the partial pressure of nitrogen in the lungs, which induces diffusion of nitrogen from the blood into the breathing gas, and this effect eventually lowers the concentration of nitrogen in the other tissues of the body. If continued for long enough, and without interruption, this provides effective protection upon exposure to low-barometric pressure environments. Most

small general aviation aircraft are not pressurized, therefore oxygen use is an FAA requirement at higher altitudes. Although pure oxygen pre-breathing is an effective method to protect against altitude DCS, it is logistically complicated and expensive for the protection of civil aviation flyers, either commercial or private. Therefore, it is currently used only by military flight crews and astronauts for protection during high-altitude and space operations. It is also used by flight test crews involved with certifying aircraft, and may also be used for high-altitude parachute jumps. Astronauts aboard the International Space Station preparing for extra-vehicular activity EVA "camp out" at low atmospheric pressure, Neurological symptoms, pulmonary symptoms, and mottled or marbled skin lesions should be treated with hyperbaric oxygen therapy if seen within 10 to 14 days of development. At a dive site, a riskier alternative is in-water recompression. It is no longer recommended to administer aspirin, unless advised to do so by medical personnel, as analgesics may mask symptoms. People should be made comfortable and placed in the supine position horizontal , or the recovery position if vomiting occurs. A US Navy treatment table 5 can be safely performed without air breaks if a built-in breathing system is not available. However, permanent long-term injury from DCS is possible. Three-month follow-ups on diving accidents reported to DAN in showed From to , they recorded 50, dives, from which 28 recompressions were required â€" although these will almost certainly contain incidents of arterial gas embolism AGE â€" a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a bubble forming in the eye of a viper subjected to a near vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised that this was the cause of death. Charles Pasley , who was involved in the recovery of the sunken warship HMS Royal George , commented that, of those having made frequent dives, "not a man escaped the repeated attacks of rheumatism and cold". First documented case of decompression sickness, reported by a mining engineer who observed pain and muscle cramps among coal miners working in mine shafts air-pressurized to keep water out. Decompression sickness reported and one resulting death of caisson workers on the Royal Albert Bridge. Bauer published outcomes of 25 paralyzed caisson workers. From to , all prominent features were established. Explanations at the time included: The Eads Bridge in St Louis employed compressed air workers including Alphonse Jaminet as the physician in charge. There were 30 seriously injured and 12 fatalities. Jaminet developed decompression sickness and his personal description was the first such recorded. The similarity between decompression sickness and iatrogenic air embolism as well as the relationship between inadequate decompression and decompression sickness was noted by Friedburg. Andrew Smith first utilized the term "caisson disease" describing cases of decompression sickness as the physician in charge during construction of the Brooklyn Bridge. Recompression treatment was not used. The project chief engineer Washington Roebling suffered from caisson disease,[23] and endured the after-effects of the disease for the rest of his life. During this project, decompression sickness became known as "The Grecian Bends" or simply "the bends" because afflicted individuals characteristically bent forward at the hips:

Chapter 9 : Uncontrolled decompression

Uncontrolled decompression is an unplanned drop in the pressure of a sealed system, such as an aircraft cabin or hyperbaric chamber, and typically results from human error, material fatigue, engineering failure, or impact, causing a pressure vessel to vent into its lower-pressure surroundings or fail to pressurize at all.

In extreme cases, symptoms may occur before the dive has been completed. Navy and Technical Diving International , a leading technical diver training organization, have published a table that indicates onset of first symptoms. The table does not differentiate between types of DCS, or types of symptom. It may happen when leaving a high-pressure environment, ascending from depth, or ascending to altitude. Ascent from depth DCS is best known as a diving disorder that affects divers having breathed gas that is at a higher pressure than the surface pressure, owing to the pressure of the surrounding water. The risk of DCS increases when diving for extended periods or at greater depth, without ascending gradually and making the decompression stops needed to slowly reduce the excess pressure of inert gases dissolved in the body. The specific risk factors are not well understood and some divers may be more susceptible than others under identical conditions. DON can develop from a single exposure to rapid decompression. Workers spending time in high-pressure atmospheric pressure conditions are at risk when they return to the lower pressure outside the caisson if the pressure surrounding them was not reduced slowly. DCS was a major factor during construction of Eads Bridge , when 15 workers died from what was then a mysterious illness, and later during construction of the Brooklyn Bridge , where it incapacitated the project leader Washington Roebling. DCS is very rare in healthy individuals who experience pressures equivalent to this altitude. However, since the pressure in the cabin is not actually maintained at sea-level pressure, there is still a risk of DCS in individuals having dived recently. Also, cabin pressurization systems still fail occasionally , and some people may be predisposed to the drop in pressure that occurs even in pressurized aircraft. The higher the altitude of exposure the greater is the risk of developing altitude DCS. They may be considered as either environmental or individual. Environmental The following environmental factors have been shown to increase the risk of DCS: Dive tables make provisions for post-dive time at surface level before flying to allow any residual excess nitrogen to outgas. Therefore, the assumption that the dive table surface interval occurs at normal atmospheric pressure is invalidated by flying during that surface interval, and an otherwise-safe dive may then exceed the dive table limits. A right-to-left shunt may allow bubbles to pass into the arterial circulation. The following individual factors have been identified as possibly contributing to increased risk of DCS: In diving, this can allow venous blood with microbubbles of inert gas to bypass the lungs, where the bubbles would otherwise be filtered out by the lung capillary system, and return directly to the arterial system including arteries to the brain, spinal cord and heart. In the brain, infarction results in stroke , and in the spinal cord it may result in paralysis. Depressurisation causes inert gases , which were dissolved under higher pressure , to come out of physical solution and form gas bubbles within the body. These bubbles produce the symptoms of decompression sickness. On ascent from a dive, inert gas comes out of solution in a process called " outgassing " or "offgassing". Under normal conditions, most offgassing occurs by gas exchange in the lungs. The formation of bubbles in the skin or joints results in milder symptoms, while large numbers of bubbles in the venous blood can cause lung damage. The most severe types of DCS interrupt â€” and ultimately damage â€” spinal cord function, leading to paralysis , sensory dysfunction, or death. In the presence of a right-to-left shunt of the heart, such as a patent foramen ovale, venous bubbles may enter the arterial system, resulting in an arterial gas embolism. Breathing gas mixtures such as trimix and heliox include helium , which can also cause decompression sickness. Helium both enters and leaves the body faster than nitrogen, so different decompression schedules are required, but, since helium does not cause narcosis , it is preferred over nitrogen in gas mixtures for deep diving. Most divers do longer decompressions, however some groups like the WKPP have been pioneering the use of shorter decompression times by including deep stops. Very deep dives have been made using hydrogen

-oxygen mixtures hydrox , [53] but controlled decompression is still required to avoid DCS. Isobaric counterdiffusion DCS can also be caused at a constant ambient pressure when switching between gas mixtures containing different proportions of inert gas. This is known as isobaric counterdiffusion , and presents a problem for very deep dives. Nitrogen diffuses into tissues 2. Switching between gas mixtures that have very different fractions of nitrogen and helium can result in "fast" tissues those tissues that have a good blood supply actually increasing their total inert gas loading. This is often found to provoke inner ear decompression sickness, as the ear seems particularly sensitive to this effect. This will result in a shorter available time under water or an increased decompression time during the subsequent dive. The total elimination of excess gas may take many hours, and tables will indicate the time at normal pressures that is required, which may be up to 18 hours. Reduction in decompression requirements can also be gained by breathing a nitrox mix during the dive, since less nitrogen will be taken into the body than during the same dive done on air. The algorithms used are designed to reduce the probability of DCS to a very low level, but do not reduce it to zero. Breathing pure oxygen significantly reduces the nitrogen loads in body tissues and, if continued without interruption, provides effective protection upon exposure to low-barometric pressure environments. Therefore, it is currently used only by military flight crews and astronauts for protection during high-altitude and space operations. It is also used by flight test crews involved with certifying aircraft. Astronauts aboard the International Space Station preparing for extra-vehicular activity EVA "camp out" at low atmospheric pressure, Neurological symptoms, pulmonary symptoms, and mottled or marbled skin lesions should be treated with hyperbaric oxygen therapy if seen within 10 to 14 days of development. At a dive site, a riskier alternative is in-water recompression. It is no longer recommended to administer aspirin, unless advised to do so by medical personnel, as analgesics may mask symptoms. People should be made comfortable and placed in the supine position horizontal , or the recovery position if vomiting occurs. However, permanent long-term injury from DCS is possible. Three-month follow-ups on diving accidents reported to DAN in showed From to , they recorded 50, dives, from which 28 recompressions were required â€” although these will almost certainly contain incidents of arterial gas embolism AGE â€” a rate of about 0. Robert Boyle demonstrated that a reduction in ambient pressure could lead to bubble formation in living tissue. This description of a viper in a vacuum was the first recorded description of decompression sickness. Giovanni Morgagni described the post mortem findings of air in cerebral circulation and surmised that this was the cause of death. Charles Pasley , who was involved in the recovery of the sunken warship HMS Royal George , commented that, of those having made frequent dives, "not a man escaped the repeated attacks of rheumatism and cold". First documented case of decompression sickness, reported by a mining engineer who observed pain and muscle cramps among coal miners working in mine shafts air-pressurized to keep water out. Bauer published outcomes of 25 paralyzed caisson workers. From to , all prominent features were established. Explanations at the time included: Alphonse Jaminet as the physician in charge. There were 30 seriously injured and 12 fatalities. Jaminet developed decompression sickness and his personal description was the first such recorded. The similarity between decompression sickness and iatrogenic air embolism as well as the relationship between inadequate decompression and decompression sickness was noted by Friedburg. He suggested that intravascular gas was released by rapid decompression and recommended: Andrew Smith first utilized the term "caisson disease" describing cases of decompression sickness as the physician in charge during construction of the Brooklyn Bridge. Recompression treatment was not used. The project chief engineer Washington Roebling suffered from caisson disease. He battled the after-effects of the disease for the rest of his life. During this project, decompression sickness became known as "The [Grecian] Bends" because afflicted individuals characteristically arched their backs: Leonard Hill used a frog model to prove that decompression causes bubbles and that recompression resolves them. The decompression chamber was invented by the Italian engineer Alberto Gianni. The US Navy published the first standardized recompression procedure. Altitude DCS is treated with hyperbaric oxygen for the first time. Robert Workman established a new method for calculation of decompression requirements M-values. The "SOS Decompression Meter", a submersible

mechanical device that simulated nitrogen uptake and release, was introduced. FC Golding et al. Orca produced the "EDGE", a personal dive computer, using a microprocessor to calculate nitrogen absorption for twelve tissue compartments. This is because scuba diving is considered an elective and "high-risk" activity and treatment for decompression sickness is expensive. A typical stay in a recompression chamber will easily cost several thousand dollars, even before emergency transportation is included.