To the disappointment of the men, Dick Alba showed little sign of further recovery or relief of symptoms. On Sunday August 9, 1981, his company sent a Lear jet to fly him at low level to a New Orleans hospital. After two-and-a-half months he was discharged and though he still walks with a stick he jokes about taking up jogging very soon. He can drive his car.

In January this year, Tony Liddicoat received a letter from Dick Alba, which says: "Due to your successful treatments of me at St George's Cay and my subsequent flight to New Orleans and admittance to JoEllen Smith Memorial Hospital, I am walking forever in your debt ..." The letter went on to invite him to be "Guest of Honor at the official opening of the new Hyperbaric Treatment Facility at the hospital on Wednesday, February 17".

The hospital had no doubt about the effectiveness of Tony's treatment. At best, without it, Dick Alba would have been a paraplegic. The hospital said that the decompression had completely removed the nitrogen from Alba's system and thus given him mobility despite the residual damage to the spinal cord.

Tony went to New Orleans and was feted "overwhelmingly ... almost embarrassing ..." But he was not embarrassed by Dick Alba's personal gift to him - an engraved Rolex helium diving watch.

In fact he looked long and hard at that watch when it was put to him that he took a risk in doing what he did. Finally he said simply: "There was no alternative, regardless of the weather and the lack of equipment - you had to do something. If I hadn't, he'd have died."

There was little doubt in anyone's mind about that. Least of all in the Army's reaction. Staff Sergeant Tony Liddicoat of the Junior Leaders Regiment, Royal Engineers, at Dover, was awarded the Queen's Commendation for Brave Conduct for his lifesaving action.

The announcement in the "London Gazette" says that his award was for "bravery, prompt action and skill when undertaking a complicated decompression programme in adverse conditions, in order to save the life of a man suffering from decompression sickness."

Which sums up why Tony Liddicoat is "Diver" magazine's Diver of the Year!

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COMMENT

SPUMS' members and other readers of this Journal will have recognised that this was a case for the Edmonds' Underwater Oxygen Therapy Apparatus. When the issue of DIVER containing this article was delivered to its readers, the SPUMS Annual Scientific Conference was discussing the treatment of decompression sickness without a chamber.

NOW READ ON...

SPUMS ANNUAL SCIENTIFIC MEETING 1982

THE RATIONALE FOR DRUG THERAPY IN DECOMPRESSION SICKNESS

AA Bove

Although decompression sickness (DCS) is a disease unique to changes in the pressure environment, data obtained over the past 20 - 25 years has led to the concept that DCS can be treated like many other serious diseases, using standard therapy developed for other purposes. Various clinical investigators, basic researchers, and a few non-medical diving engineers and supervisors conceived of recompression therapy for "diver's rheumatism" in the latter part of the 19th century. Recompression was probably first used for the treatment of caisson workers. Organized decompression and treatment tables for air appeared in the late 19th and early 20th centuries. Oxygen for decompression and for the treatment of DES appeared in the 1920-1935 period, and until recently, recompression and oxygen were the only means for treating DES. From the 1930's to the present time, a body of knowledge slowly developed which culminated in the understanding of DES as a process which triggers body wide inflammation, which can be treated like any other disease process that activates the inflammatory process.

Based on these newly developed approaches to the treatment of DES, one can outline a treatment strategy which, of necessity, begins with recompression and oxygen, but which also includes fluid therapy, the use of corticosteroids, consideration of anti-coagulants, the use of anti-platelet agents and of non-steroidal anti-inflammatory agents such as indomethacine or ibuprofen.

It is clear that the bubble-triggered inflammation which occurs in DES is not reversed by recompression, and although the ischaemic component is resisted by hyperbaric oxygen, use of oxygen and recompression will not halt the inflammatory responses already underway. The process of inflammation encompasses a collection of responses which act normally to counter invasion of the body by foreign organisms (eg. bacteria, viruses, parasites). To gain insight into the use of drugs in DES therapy, we will examine the components of inflammation and the drugs which affect them.

Permeability

The vascular system retains fluid by holding protein molecules in the blood vessels. The osmotic effect of the protein holds water in the vascular compartment. Occasionally, it is expedient for proteins to leave the plasma and enter the tissue spaces outside the blood vessels. The most important need is when antibody diffuses into tissues invaded by bacteria or other organisms. To move protein from the vascular system, a gating mechanism is present in the post-capillary venule. The endothelium in this region can contract, separate cell junctions and allow protein to leave the vascular system. Control of the gating mechanism is through the

inflammatory process. Agents such as histamine, serotonin, bradykinin and certain prostaglandins stimulate contraction of post-capillary venular endothelium and cause an increase in permeability. Direct vascular injury with loss of normal endothelial integrity also increases permeability. When inflammation is triggered locally, by local injury, infection, etc, the reactions which occur locally cause release of vasoactive mediators, protein leakage locally and the mounting of a local attack against the invading organism. The local effect is a normal, necessary, response for protection against a wide variety of organisms. Consider now, how DCS will interact in this process. We have shown in a series of experimental studies that the presence of bubble surfaces in the blood activates Hageman factor, which in turn triggers the clotting cascade, and the complement activation chain. The same processes activate kinins and fibrinolysins. The complement chain produces substances which ultimately release histamine from most cells, serotonin from blood platelets, and activate kinins and prostaglandins. These reactions occur bodywide. There is no localization. Thus protein and water (plasma) leak from the bloodstream into tissues everywhere. Lung oedema is one consequence of this reaction in DCS, and significant arterial desaturation will occur when lung oedema is severe enough to compromise oxygen transport. The massive plasma leak also reduces blood return, haemoconcentrates the blood and will produce hypovolemic shock when severe enough. The entire reaction, triggered by bubble surfaces in the blood, can become self sustaining when tissue injury further activates inflammation. Thus recompression will not reverse this process and intervention with drugs is necessary.

Histamine release stimulates the endothelial cells which contract and open up little spaces between them which is a physiological response. In decompression sickness histamine release occurs all over the circulation. In addition white cells are activated by a number of things. Usually the beginning of the process is a bubble Hageman factor interaction. Hageman factor activates complement, then one of the products of complement activates the white cells. In other words, the signal goes to the white cells saying that there is an inflammatory process going on in the body. The white cells develop pseudopods and dig their way through the endothelium in the area where injury or inflammation has occurred. There are chemotatic proteins that tell the white cells where to go. You get an injury to a tissue which releases a chemotactic material. There is Hageman activation. The white cells are told to go to that place and leave the vascular system and go out and attack something. But in decompression sickness, the chemotactic compounds are distributed all over the vascular system, so that the white cell is getting a signal to dig through the epithelium everywhere. That is a different process to the physiological process. The white cell will damage the vessel wall and that is a component of the increased permeability. Another is the histamine reaction which is a physiological one. Histamine combined with direct injury gives a more prolonged permeability response.

In decompression sickness we have the physiological responses to injury in the micro-circulation, but it is a larger diffuse reaction than usual. Not just a local reaction, but the whole micro-circulation is stimulated. In addition to that we get vessel endothelium damage in many places, because the white cells are being told to go to work. So you get a response which includes the histamine response, and the direct vascular injury response. It is the direct injury response that damages endothelium and causes the more prolonged leakage.

We would like to treat the histamine induced response, which is the physiological response. We can block that, with steroids for example. At the same time we want to tell the white cells to stop digging up the endothelium. You can do that with steroids as well. Once that has been done, the histamine reaction will stop quite soon. The direct damage will not go away. It will have to heal and it takes several days for that to occur. When you are dealing with permeability changes you want to stop the process and replace the fluids. Then keep the fluids balanced until the system heals itself. Direct injury response may take twenty four to thirty six hours to recover. The histamine permeability response, if you stop it, can revert in half an hour. They are two different mechanism.

Clotting

Two different systems are involved with blood clotting in DCS. The intrinsic clotting system, triggered by surface activation of Hageman factor produces thrombi at gas-blood interfaces. When significant bubbling occurs in the blood (eg. in severe DCS) massive clotting can occur. Our previous work indicates that thrombosis of the vertebral venous plexus contributes to spinal DCS. When DCS occurs following blowup, it is likely that massive blood clotting contributes to the high mortality which these accidents produce. The second component of clotting which must be considered is the blood platelet. Bubble surfaces also activate platelets causing them to aggregate and form platelet thrombi on vessel walls and in the microcirculation. Besides the occlusive properties of platelet embolic or local thrombi, platelets release several vasoactive compounds which increase permeability (eg. serotonin) and cause vasoconstriction (eg. thromboxane). Platelet released thromboxane not only causes vaso-constriction, but also increases permeability, further stimulates platelet aggregation, and may activate leucocytes to migrate through vascular endothelium, causing further damage to the blood vessel walls. Once again, recompression will not reverse this process, once it has begun, and drugs which specifically interfere with these inflammatory processes are necessary for treatment.

If you look at clotting in the presence of bubbles, you will activation of the clotting system. That can be in the plasma (intrinsic) and with platelets. There are two different components. The platelet is directly activated by the bubble surface. It releases platelet factor 4 and activates

clotting. You can still activate clotting without platelets. The only protein in the clotting chain that is sensitive to surfaces is Hageman factor. It is sensitive to the lipid monolayer formed around the bubble. The centre of some of the lipo-proteins in the blood become denatured and adhere to the bubble and change the polarity so that there is a lipid monolayer around the bubble surface. That may be the link that gets Hageman factor activated. The fact is that you will find intrinsic clotting activated by bubbles and as far as we know the only component of the clotting system that is sensitive to bubble is Hageman factor. It may not be the surface per se which causes it, it may be the lipid layer that forms from the lipoproteins around the bubble.

These briefly described processes can be altered by agents which interfere with one or more of the components of the inflammatory reaction which has been triggered by bubbles in the blood or by tissue injury caused by gas expansion or by local ischaemia.

THE TREATMENT OF DECOMPRESSION SICKNESS

AA Bove

The treatment of the diver or aviator with decompression sickness (DCS) should always be based on recompression therapy. Recompression is the most important modality, but for the best results it should be combined with oxygen, adequate fluid therapy, corticosteroids, other anti-inflammatory drugs and, in selected cases, anticoagulants. When no chamber is readily available, the other measures used for therapy, even when successful, must be considered as inferior or palliative. Recompression treatment should always be provided even if a significant delay is anticipated prior to recompression.

RECOMPRESSION

Standard recompression therapy can still be based on the somewhat artificial, but practical division of DCS into minor and major types. Minor DCS involving joints is treated with a US Navy table 5 or equivalent [60 ft (18m) intermittent air/O_2] lasting two to two and a half hours. Major DCS is treated with a US Navy table 6 or equivalent [60 ft (18m) intermittent air/O_2] lasting about five hours. Other useful tables such as the Comex 30 [100 ft (3Om) intermittent air/O_2 and air/50% Nitrox] should be made available when a deeper treatment depth is required, and saturation is impractical. Special adaptations of the 60 foot oxygen table can be made for a monoplace chamber where pure oxygen is used to compress the chamber. In-water recompression can used with extreme caution and careful planning, when nothing else is available and the diver's life is threatened.

OXYGEN

The next most important treatment is oxygen. The use of 100% oxygen at 60 ft, 50% oxygen at 1OO ft and 100% oxygen at 1 ATA have all been established as useful therapy for DCS. Two benefits accrue from oxygen: ischaemic tissues may be provided with enough O2 to prevent cell death, and inert gas will be removed from bubbles and replaced by oxygen which will be metabolized. Although administration of oxygen appears simple, several precautions should be mentioned. The oxygen must be supplied with a tight fitting mask to ensure that the necessary concentration is provided. An overboard dump must be used in multiplace chambers to prevent oxygen build up in the chamber atmosphere. The hazard of fire with high pressure oxygen must be borne in mind. Oxygen should be provided to divers with DCS, while at one ATA awaiting transportation and during transit to a treatment chamber. Oxygen toxicity will not be a problem at one ATA unless oxygen breathing is prolonged. (eg. four or more hours continuously). At 60 ft on 100% oxygen however, signs of CNS oxygen toxicity must be watched for diligently.

FLUIDS

Fluid therapy is the next treatment consideration. The diver with DCS is likely to be hypovolaemic because of plasma loss into tissues, and may be dehydrated because of inadequate fluid intake, excess fluid loss or vomiting. The resulting hypovolaemia and haemoconcentration can aggravate the microcirculatory perfusion defect associated with DCS, and produce permanent tissue injury. Thus an important goal of therapy in DCS is to prevent haemoconcentration and hypovolaemia. The best replacement fluid is a crystalloid solution such as normal saline. The use of plasma or dextran does not appear to provide additional benefit. Also these solutions can result in fluid overload which is not readily corrected. Conversely, an overload of crystalloid is easily dealt with by giving a diuretic. Enough fluids should be given to maintain urine output at 100 cc/hour. Calculations based on the standard estimator for fluid deficit can be used successfully. Be sure the patient has normal lower urinary tract function, since bladder paralysis from cord injury may result in urinary retention and an improperly estimated fluid need. One should start an intravenous line in divers with DCS whenever practical and infuse normal saline to reverse or prevent hypovolaemia. One should monitor the urine output and blood pressure. It is essential that the state of bladder function is known.

CORTICOSTEROIDS

Corticosteroids should also be used in treating serious DCS. The glucocorticoids interfere with the inflammatory process. In DCS where the inflammatory reaction is inappropriate and detrimental to the diver, aborting