

Aspirin and Decompression Diving

The following text taken from internet is quite illuminating. It explains how Aspirin and related drugs affect blood chemistry and the possible effects on divers.

As for ibuprofen while diving, 800 mg every 4 hours or even as a pre-dive ritual seems excessive, as you probably only need one 200 mg tablet in a 24-hour period. God almighty you guys must bleed like hell if you ever cut yourself (ibuprofen prolongs bleeding time just like aspirin, not to mention the effects it has on your stomach).

It's next to impossible to give any hard line "best effect dose" which attains the best of ibuprofins benefits with the least amount of side affects. I would submit that it's part science, and part 'art' on behalf of the diver. High doses are definitely required to gain benefit mind you, and it would appear that at least a few others on this list are unaware of its use and benefit. For the moment, I wish to address aspirin specifically and not ibuprofen. Although both are used for the same purpose, aspirin's use has been widely documented with respect to decompression diving. I submit the following to whomever for the sake of further discussion and intelligent hypothesis.

Aspirin is a powerful medication and is actually an analgesic and an anti-inflammatory drug. Aspirin is a brand name in Canada; acetylsalicylic acid is the generic name. ASA, a commonly used designation for aspirin (or acetylsalicylic acid) in both the U.S. and Canada, is the term used in Canadian product labeling. Aspirin is an over-the-counter (OTC) medicine, and because it is so common and so readily available, many people do not consider it a "real medication." This is a common misconception and aspirin is a very "real drug." Its use in staged decompression diving has been extrapolated from other benefits that aspirin has been prescribed for (1). Aspirin's main use in diving is to prevent blood clotting and platelet aggregation. Although aspirin is referred to as a "blood thinner," it does not actually "thin" the blood. Instead, anticoagulants alter proteins in the blood that are responsible for clotting while antiplatelet drugs prevent platelets from clumping and forming clots. Aspirin functions to make the platelets less 'sticky', thereby acting as an anti-coagulant. Aspirin is an aid; not a substitute for proper hydration, even though its main benefit to the diver is to allow improved blood flow and gas transport by increasing tissue gas perfusion. In vitro and in vivo studies have shown that hyperbaric pressure increases red blood cell (RBC) aggregation (2). Enhanced RBC aggregation in pathologic states can cause increased clotting. Both aggregation and clotting hamper the transport of gas which may lead to any number of hyperbaric related injuries.

It is known that the hyper-aggregability of platelets is remarkably important in the pathogenesis of decompression sickness (3). One investigation (2) examined the effects of pressure on RBC aggregation in human volunteers. The hypothesis tested was that RBC aggregation is increased during hyperbaric exposure. Subjects participated in dives to 300 fsw in a chamber. Blood samples were taken at the surface, at 66 fsw, and at 300 fsw. The median aggregate size (number of RBC/aggregate) of RBCs was significantly increased at depth. The deeper one goes, the greater the aggregate size. These results show that even mild pressure increases RBC aggregation in the human circulation. Therefore, aspirin is used as a preventive measure to a known prohibitor of gas transport, which may lead to symptomatic DCS. There are some controversial lines within the diving community concerning the use of aspirin. All groups are aware of the later; the segregation comes from

discussion of aspirin's effect on blood viscosity. There are some who contend that aspirin will reduce blood viscosity and therefore do more harm than good. Reduced blood viscosity would reduce gas tensions and therefore contribute to micro bubble formation. It is unproven however, that aspirin will decrease the viscosity of blood and contribute to micro bubble formation.

Decreases in systemic hematocrit (blood count of red cells) tend to decrease blood viscosity and promote microvascular vasomotion and tissue perfusion (4,5), whereas an abnormally high hematocrit increases blood viscosity and results in clumping and aggregation of the erythrocytes, capillary occlusion and regional redistribution of the circulation. One study (6) examined the effects of aspirin and dipyridamole (pronounced dye-peer-id-a-mole -- its a powerful platelet aggregation inhibitor; antithrombotic adjunct) on platelet function, hematology, and blood chemistry of saturation divers. 24 divers were assigned randomly to 4 treatment groups. Group I received aspirin (325 mg) t.i.d. (ter in die, Latin meaning 3 times a day); Group II received dipyridamole (75 mg) t.i.d.; and Group III received both drug regimens; while group IV received matching placebo. Double-blind procedures were followed. Treatment began 24-h prior to a 48-h saturation dive (inclusive of 17 hour decompression) and continued throughout and for 3 days after the dive. A post-dive reduction in circulating platelet count was observed in all groups, except the group that received aspirin only. Platelet survival was shortened in all treatment groups. Five cases of Type I DCS occurred and were treated by recompression, two in the aspirin plus dipyridamole group, two in the dipyridamole group, and one in the placebo group; none in the aspirin only group.

Blood chemistry and hematology profiles showed that divers with decompression sickness had elevated GOT (glutamic oxaloacetic transaminase), GPT (glutamic pyruvic transaminase), and CPK (creatinine phosphokinase is one of several chemicals usually released in the blood after a heart attack, an increase of this form of isoenzyme in the blood is a diagnostic clue to tissue damage). Divers with DCS had more elevated cholesterol and triglyceride levels, and greater reductions in platelet count, platelet factor 4 and thrombin (an enzyme formed in the clotting) clotting time than most other subjects. Subjects receiving either aspirin or aspirin plus dipyridamole had fewer changes in these parameters. Failure of aspirin to potentiate, or add to, dipyridamole may be due to other actions of aspirin such as inhibition of prostacyclin (PGI₂) synthesis. (PGI₂, a prostaglandin, is formed mainly in the blood vessel walls and slows blood platelet clumping. Aspirin, in doses as little as 4 mg/kg of body weight, inhibits prostacyclin as well as thromboxane formation. Prostaglandins may induce or inhibit platelet aggregation and constrict or dilate blood vessels. For an in-depth overview on prostaglandin and thromboxane biosynthesis; the role of steroidal and non-steroidal anti-inflammatory drugs; the reader is referred to an excellent review by Smith et al (7))

This particular study (6) seems to favour the use of aspirin in a hyperbaric environment, however further studies of the role of antiplatelet drugs such as dipyridamole in decompression sickness may be warranted. These results indicate that the combination of aspirin and dipyridamole offers no measurable advantage over aspirin alone. This study also suggests that antiplatelet drugs such as dipyridamole may actually be a contraindication for a hyperbaric environment. Yet another study examined the hematology and blood chemistry in saturation diving using antiplatelet drugs, aspirin, and VK744. Blood chemistry and cellular parameters were studied before, during, and after saturation dives in a habitat, on two separate occasions. The results confirm previous observations and indicate that post-decompression loss of platelets may be related to sequestering of reactive platelets, possibly by microbubbles, and that the phenomenon can be inhibited by some antiplatelet drugs. Lastly, it should be stated that in vitro and in vivo research clearly demonstrates the influence of nutrition on platelet aggregation and clumping ie. eating fatty foods compounds the problematic blood chemistry situation (8-11).

Aspirin is effectively used by many staged decompression divers who can tolerate the

drugs side effects. In general, sustained release doses by divers, range from 325 mg to 600 mg, (single one time dose) taken 60 to 120 minutes before a dive. There does not appear to be a specific or "magic" dose to provide for the best protection with the least amount of side effects. The anti-aggregating therapy usually associated with hyperbaric treatment involves administration of acetylsalicylic acid in low doses; 3.5 ~ 5 mg/kg of body weight (3). During one study (12), platelet functions were studied after various single doses of aspirin (75 mg, 150 mg, 300 mg, and 600 mg) in 20 males. Clotting time and platelet counts remained unchanged. Significant de-aggregation of platelets occurred only with 600 mg of aspirin. Another study (13) by Heavey et al, reports that an oral dose of aspirin (600 mg) causes rapid and substantial inhibition of bradykinin-stimulated PGI₂ production, but recovery occurs within 6 hours; this implies that endothelial PGI₂ synthesis would be spared most of the time during dosing once daily with even this relatively large dose of aspirin (13). Yet another study (14), examined the effect of chronic administration of variable low doses of aspirin on platelet adhesiveness, platelet count, bleeding time and clotting time to find out, as to how low the dose of aspirin needs to be in order to have an effective antiplatelet effect in individuals who require such therapy (meaning over a longer period of time).

A statistically significant reduction in the platelet adhesiveness was observed in all the groups, but the best effect was exhibited by 50 mg of aspirin dose. Bleeding time was also increased in all the groups but statistically significant difference were observed with 50, 75 and 100 mg doses. So far we have doses somewhere between 50 mg/day, minimum for long term chronic dosing; 325 mg t.i.d. for up to 5 days dosing (15); to 600 mg/day one time minimum effective dosage. If one cares to search, they will find a myriad of studies for aspirin and effective dosages. Therefore it is next to impossible to give any hard line "best effect dose" which attains the best of aspirin's benefits with the least amount of aspirin's side effects. There are several brands of coated aspirin such as 'Entrophen 10', an enteric coated tablet of ASA, which are dissolved in the gut instead of the stomach (650 mg effective for up to six hours or so). What is known however, is that antacids can decrease the effectiveness of aspirin. Since aspirin is an analgesic and an anti-inflammatory, where high doses are used, it may mask mild symptoms of DCS. Many antihistamines and corticosteroids used by divers for certain conditions, to aid in ease of equalization, can have the same effect. Excessive bleeding may also be a concern from an acquired injury such as cuts, bruises, broken bones etc. Bleeding into the middle ear or sinus from a squeeze may require special precaution as well. Every diver has minor trauma that is usually of little consequence. This can become a major problem if the diver is on prescription anticoagulants, however most authorities (Bove, Davis, DAN, etc.) agree that divers taking coumadin or other anticoagulants is either a relative contra-indication or an absolute contraindication to diving and therefore not an issue (16).

As well, aspirin may have more benefits to the decompression diver, with less side effects than those of anticoagulant drugs such as coumadin, dipyridamole, heparin etc. The added side bonus of aspirin in deep diving is of course, that it helps prevent pain associated with CO₂ headaches commonly attributed to hard work and/or improper breathing techniques underwater. In short, headache is a sign that something is not right, however it's not a sure sign of CO₂ buildup. The need for proper, slow, moderate-sized deep breathing technique during extreme depth diving cannot be overstated mind you.

Randy F. Milak (1)

Popovic P, et al. Levodopa and aspirin pretreatment beneficial in experimental decompression sickness. Proc Soc Exp Biol Med. 1982 Jan;169(1):140-3. (2)

Taylor WF., Chen S, Barshtein G, Hyde DE, Yedgar S. Enhanced aggregability of human red blood cells by diving. Undersea Hyper Med 1998; 25(3)167-170. (3)

Reggiani E, et al. Blood coagulation processes in decompression sickness and hyperbaric therapy. Minerva Med. 1981 May 31;72(22):1383-90. (4)

Messmer K. Blood rheology factors and capillary blood flow, in Gutierrez G, Vincent JL (eds). Update in Intensive Care and Emergency Medicine, Vol 12, Tissue Oxygen Utilization. New York, Springer-Verlag, 1991, pp 103-113. (5)

Restorff WV, Hofling B, Holtz J, et al. Effect of increased blood fluidity through hemodilution on general circulation at rest and during exercise in dogs. Pflugers Arch 1975; 357: 25-34. (6)

Philp RB, Bennett PB, Andersen JC, Fields GN, McIntyre BA, Francey I, Briner W. Effects of aspirin and dipyridamole on platelet function, hematology, and blood chemistry of saturation divers. Undersea Biomed Res 1979 Jun;6(2):127-46 (7)

Smith WL, et al. Prostaglandin and thromboxane biosynthesis. Pharmacol Ther. 1991;49(3):153-79. Review. (8)

Adam O, et al. Platelet aggregation and prostaglandin turnover in man during defined linoleic acid supply with formula diets. Res Exp Med (Berl). 1980;177(3):227-35. (9)

Temme EH, et al. Individual saturated fatty acids and effects on whole blood aggregation in vitro. Eur J Clin Nutr. 1998 Oct; 52 (10)

Gathered by Mark Ellyatt