Decompression Sickness (DCS) is caused by inert gas bubble formation in blood vessels and tissues resulting from super saturation during inadequate decompression. 'Diving acclimatization' refers to a phenomenon that occurs when individuals undergoing repeated compression – decompression cycles are able to reduce their susceptibility to DCS. The present study examined whether intensive scuba diving and physical training would reduce bubble formation and modulate endothelial function. For this purpose 22 military divers ages 25.3+/- 0.8 yrs, diving experience 46+/-3 dives during last 5 yrs, were taken. None had any episode of DCS in past. They were studied before and after a 3 months program comprising of physical training and open - sea air diving (67 dives). Skin blood flow was measured using laser Doppler flowmetry in forearm, at fixed site in 3 conditions at 1) rest- baseline 2) reactive hyperemia–endothelial dependent vasodilatation which in turn depends on NO formation 3) local heating – maximum vasodilatation and is endothelium independent. Pulse Doppler was used to see for venous bubbles. During training no diver had DCS. Kisman Integrated Severity Score (KISS) was calculated as a measure of post- decompression intravascular bubble activity status. They were significantly decreased immediately after diving training period and increased 3 months after this period. No significant difference was noticed in cutaneous vascular conductance in pre and post diving values both in reactive hyperemia and reponse to heating as compared to baseline values. Repeated Scuba diving and regular physical exercise is seen to reduce bubble formation and probably have a protected effect against DCS risk. The present study was done with the hypothesis that increased frequency of repeated compression-decompression cycle exposures would reduce circulating bubble level and modulate endothelial function by improving NO production. Physical exercise training is seen to be beneficial against type-II DCS. The main mechanism of this could be due to NO production. Here endothelial function of skin circulation was investigated as a guide to NO formation. However in the present study no such inference about the effect of NO function related to bubble formation could be made. Other biochemical mechanism may be responsible for the protective effect on bubble formation. Decompression may affect vasculature in different ways according to the type and location of vessel. Other mechanisms for diving acclimation could be depletion of complement proteins, accumulation of protective factors like Heat Shock proteins(HSP), possible linking of HSP and endogenous NO pathway or bubble formation could have activated stress response responsible for diving acclimatization. Phenomenon of repeated scuba diving and physical activity preventing against DCS is known for decades, however the mechanism remains complex. Present study could not conclude any mechanism. The mechanism underlying this adaptation needs to be elucidated.

Modern methods of G protection make pilots relatively resistant to G-LOC. But there is another G-related threat that needs attention, called G-induced vestibular dysfunction (GIVD). Pilots who experience high positive and negative G loads during unlimited aerobatic competitions and air-shows demonstrations may manifest an unstable gait after landing and walking from their aircraft, which they call the “wobbles”. An unofficial survey taken during the world aerobatic championship revealed that more than 75% of team members experienced at least one episode of GIVD. Several cases of vertigo, disequilibrium, and spatial disorientation after or during G load exposures have been reported. Animal studies have shown displacement, degeneration and damage to otocnia after various high G exposures. But yet, well controlled human studies are not reported. This study was conducted to explore GIVD under controlled conditions. 11 male military pilots who had no history of vertigo or neurological disturbances were exposed to +9 Gz for 10 seconds in a centrifuge with an onset rate of 1 G per second. The subjects wore anti G suit and performed M1 straining maneuver during the runs. Vestibular function tests, which included semicircular and otolith function tests were performed within 20 min before and after the centrifugation. The authors found that no subject had signs or symptoms of GIVD and statistics showed no difference between all vestibular function tests before and after +9Gz exposure. The clinical picture of GIVD is consistent with vertigo, but diagnosed often as Benign paroxysmal positional vertigo (BPPV) by ENT specialists. GIVD is determined by shearing forces to otolith, but the forces are of low magnitude and longer duration in contrast to sudden blunt force from head trauma in BPPV. GIVD occurs when this shearing force overcomes the conjugation force between the otocnia and gelatin layer.

The authors explain that the reported high incidence of GIVD could be related to poor conjunctions between the otocnia and the gelatin layer, which often can be caused by pathological factors or individual differences. Head movement during high G exposure also plays a role in amplifying the effect of shearing force of G load to the maculae. The negative results in this study could be due head held straight during high G and also in this study, the pilots were relatively younger with shorter flying experience whereas most reported cases were subjects of aerobatics and were older and BPPV is common in older subjects. There are also differences between in-flight and centrifuge G exposure which could cause a different stimulus to the semicircular canals contributing to incidence of GIVD. The authors conclude that though this study found no GIVD and no significant vestibular function changes in pilots after high G exposure on a centrifuge, the effects of G loads in different directions, head movements, etc on GIVD in actual flight need to studied in future in view of next generation agile aircrafts.