Long-term high +Gz effects on cardiac functions in the pilots

Jet pilots in military aviation are exposed to greater amounts of and longer duration of acceleration forces as compared with transport/helicopter pilots. Of these acceleration forces, + Gz is the most influential factor during flight. In the article published in the current issue of the Anatolian Journal of Cardiology, Öztürk et al. (1) may be of interest with its aim to determine long-term cardiac effects of this factor.

The jet and transport/helicopter pilots are exposed to various conditions, such as high altitude, low atmospheric pressure, acceleration, flight maneuvers at various durations and intensities. The respiratory rate and heart rate are susceptible to increases in changes in the atmospheric pressure and the G force in the jet pilots. The transport/helicopter pilots are not exposed to the G force because they fly below 15,000 feet; but, unlike jet pilots, they are subjected to extreme vibration. Even though supplemental oxygen is not needed and the cabin pressure is not regulated, helicopter pilots still have to deal with the effect of high altitude.

Pressure changes and the G force are among the important factors for jet pilots. The direction in which the accelerative force is exerted to the body is important in determining the effects of acceleration. The inertial force, also known as the G-force, is the force produced by the reaction of the body to an accelerating force, equal in magnitude and opposite in direction. As a result of toe to head acceleration, the inertial force or gravitational force results in a head to toe positive G force. For example, a person who weighs 70 kg (1G force) is exposed to 9x70=630 kg of force under + 9 G force. If acceleration is in the reverse direction then a negative G force is applied on the pilot's body. Jet pilots are usually exposed to a positive G force, and the blood flow to the brain, lungs and heart decreases, while that to the lower extremities increases. The anti-G straining maneuver, positive-pressure breathing and various anti-G suits are used in order to decrease the G force and hypoxic effects of flight.

There are few studies with inconclusive results regarding the effect of acceleration on the cardiac dimensions and function of high-G aviators (HGAs). Carter et al. (2) showed no evidence of structural or functional cardiac changes in the aviators exposed to high positive G forces. An increase in the right ventricular dimensions in HGAs, compared to the transport aviators, was reported in another study (3). This finding was concordant with the elevated right ventricular pressure in miniature swine exposed to acceleration stresses (4). Data from these studies are not adequate to clarify long-term high +Gz effects especially on cardiac diastolic and systolic functions of pilots.

Study by Öztürk et al. (1) is quite valuable in this regard; and it has been observed that long-term severe + Gz exposure has no effect on the anatomic structure, and systolic functions of the heart; however, it affects right ventricular diastolic function. This effect has been attributed to chronic + Gz adaptation or high levels of pulmonary artery pressure. Both exposures to +Gz and anti-G measures result in repetitive intrathoracic hydrostatic changes, which can cause significant changes in cardiac preload and afterload. In addition, under high +Gz conditions, circulatory system is mostly affected. The right ventricular wall is thinner, and it is more easily affected by changes in pressure and volume. The conditions where high +Gz occurs may have influenced the right diastolic function. The authors might have included a third group comprising of individuals who had nothing to do with aviation, for helicopter pilots are exposed to altitude hypoxia, affecting especially the right ventricle and pulmonary artery pressure.

References

2. Carter D, Prokupetz A, Harpaz D, Barenboim E. Effects of repeated exposure to acceleration forces (+Gz) and anti-G maneuvers on cardiac dimensions and performance. Exp Clin Cardiol 2010; 15: e10-e2.