Use of a hyperbaric chamber to produce simulated G-induced arm pain

Dr UI Balldin, Dr LP Krock

Crew Technology Division, Armstrong Laboratory, Brooks Air Force Base, Texas and National Defence Research Establishment, Sweden

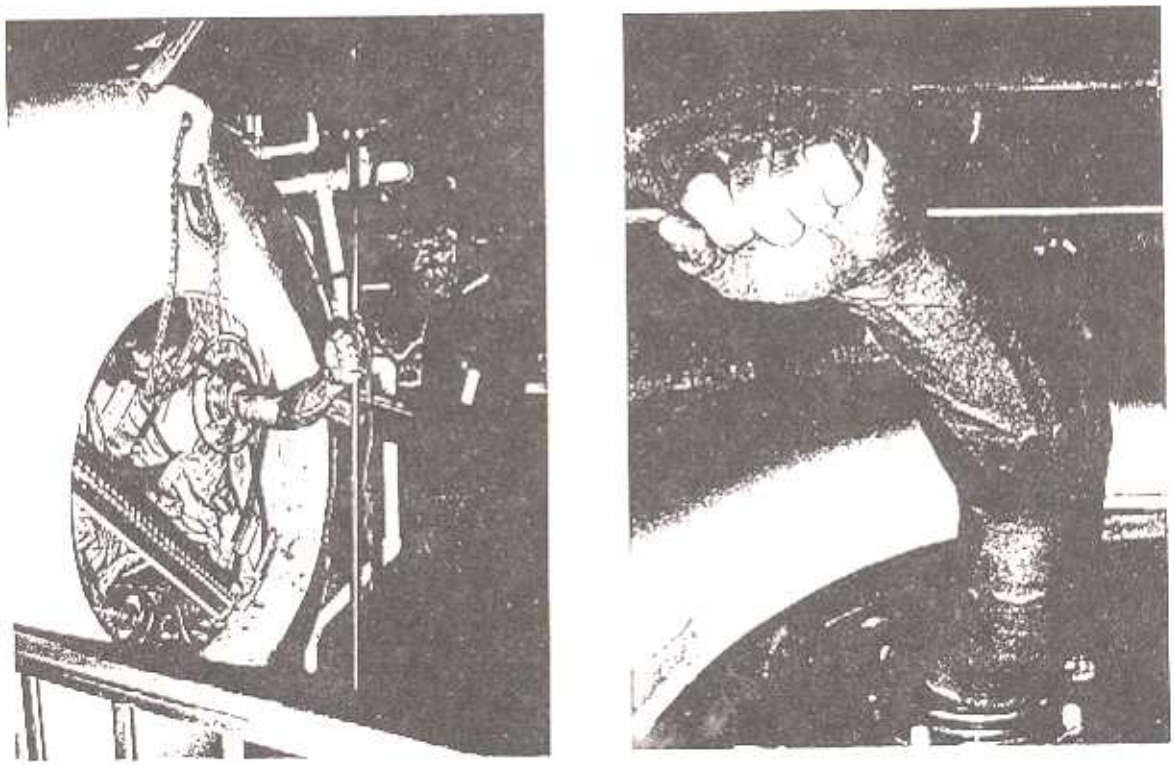
An experimental model has been developed to provoke arm pain by exposing subjects to increased ambient pressure in a hyperbaric chamber with an arm extended through a special opening and maintained at normal atmospheric pressure. Blood is forced out into the arm, causing venous congestion and ultimately arm pain. Sealing the arm in the chamber door was accomplished by an adjustable 'camera iris' opening that did not interfere with blood flow. Regional volumes of the arm were determined by impedance plethysmography. Blood vessel size and blood flow were determined by sonography. Finger blood pressure was determined by Finapres. Preliminary results indicate that with a 100 mmHg pressure increase in the chamber arm pain was provoked after 5–10 min. With higher pressure arm pain was very strong and developed earlier. The arm pain induced using this method is similar to that encountered during high G loads with pressure breathing and low hand position. This is an excellent method to simulate and study G-induced arm pain; it provides better physiological data than centrifuge-induced arm pain and countermeasures to reduce arm pain are easier to test.

Keywords: G stress; Arm pain; Hyperbaric chamber.

Exposure to high +Gz forces may cause petechiae and pain, especially around the ankle and feet of fighter pilots [1]. Increased hydrostatic pressure, with increased transmural pressure, causes overdistension of dependent blood vessels, nerve stretching and compression of skeletal muscle, with loss of plasma volume to extravascular tissue, and the occurrence of pain. These conditions can be offset by direct tissue pressurization with an anti-G suit when equipped with pressure socks. Mechanisms potentially causing this discomfort in the unprotected state are: (1) constriction of vascular smooth muscle due to myogenic and/or neurogenic responses from the distending stimuli; this response is more pronounced on the arterial side; (2) increased extravascular pressure due to skeletal muscle contraction from the anti-G straining measures, and (3) increased venous pressure in the extremities from intravascular hydrostatic pressure from the high-G exposure.

G-induced arm pain and petechiae are experienced by centrifuge subjects or pilots during extended or repeated periods of high G loads [2–4]. The pain and petechiae usually occur when the control stick and throttle, and thus the hands and the arms, are positioned below the heart level. For the pilot sitting in a standard ejection seat, the hand may be in the worst possible position, about 25 cm below the heart level. This position may create high arterial pressures in the lower arm at 9 +Gz with venous pressures increasing in similar magnitude to up to 170 mmHg according to Prior and Tozer [2]. This high intravascular pressure needs a mechanism to support vessel integrity. The compliant veins are more vulnerable to high intravascular pressures than the arteries due to their thin wall with a large lumen, relative shortage of vascular smooth muscle and a sparse vasoconstrictor innervation. The resulting arm pain appears to be the result of an overdistension of blood vessels, mainly the large systemic veins in the lower 2/3rd of the upper arm.

With the introduction of balanced pressure breathing during G (PBG), with counterpressure to the thorax, the venous pressure in the arms is increased. The result is a tendency for more arm



Figures 1. Experimental set-up for provoking arm pain.

pain and petechiae in the elbow region during high sustained G [2]. In this area the blood vessels are superficial and without tissue counterpressure.

Prior and Tozer [2], in measurements of forearm venous pressure at increased G loads, found that arm pain developed when venous pressure in the arm reached 150 mmHg. Above that pressure, arm pain directly correlated with venous pressure. Also, the incidence of arm pain increased with the application of PBG, with (balanced) or without counterpressure to the chest.

Applying a moderately increased pressure directly to the affected area of the arm during high G loads is likely to reduce the pain and the development of capillary leakage.

Provocation of vessel overdistension and its resultant arm pain also may be accomplished by exposing a subject to a hyperbaric pressure in a chamber with an arm extended through the wall that is exposed to atmospheric pressure. This method of inducing differential pressures in the arm provides a model for studying the physiologic mechanisms involving G-induced arm pain that is more convenient than using the centrifuge.

Methods

An experimental (venous congestion) model has been developed provoking arm pain by exposing seated subjects to a hyperbaric pressure in a chamber with an arm at atmospheric pressure extended through an opening in the chamber (Figure 1). The model has been used on 3 subjects. We hypothesize that blood will be forced into the arm and with less venous return venous

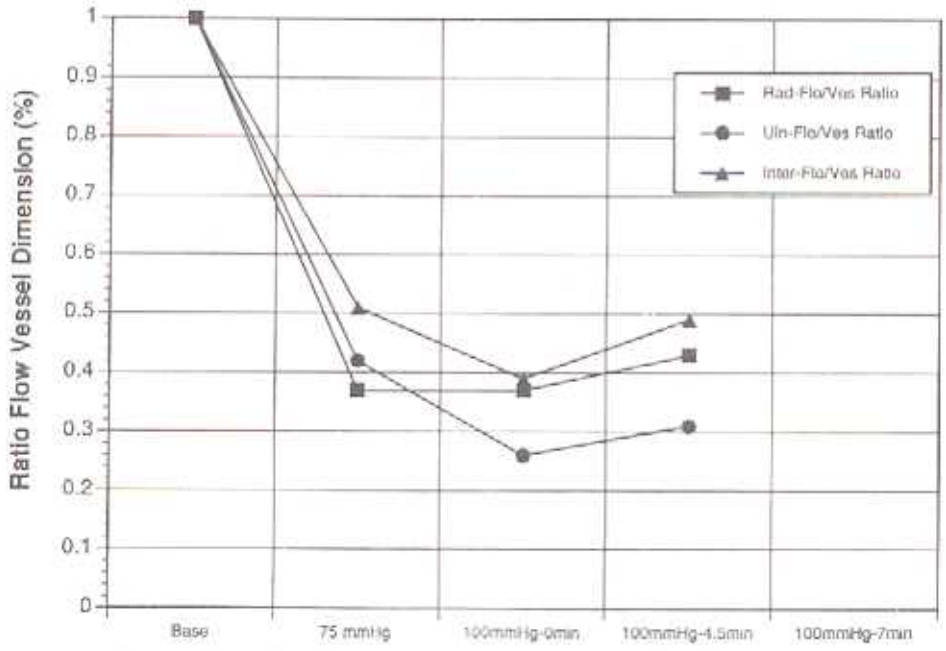


Figure 2. Blood flow/vessel dimension for radial, ulnar and interosseos arteries.

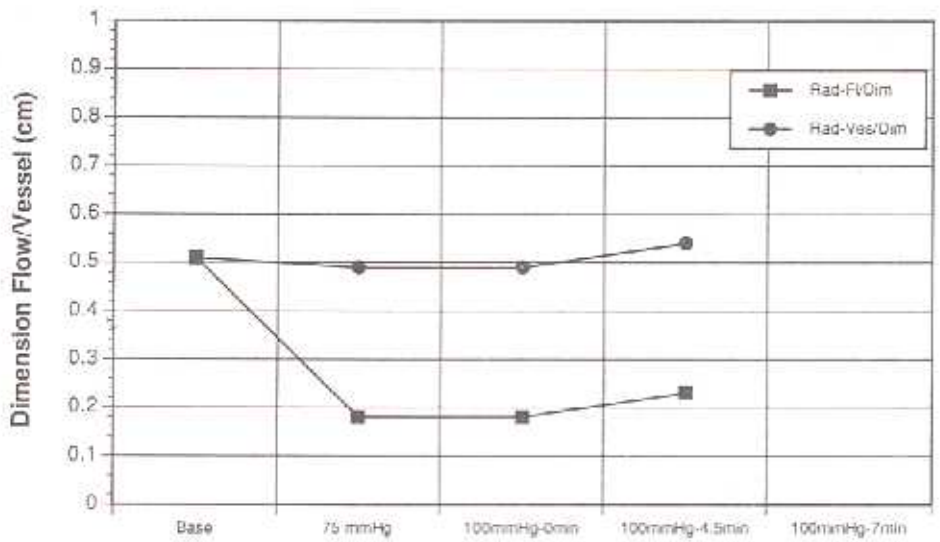


Figure 3. Blood flow and vessel dimension for radial artery

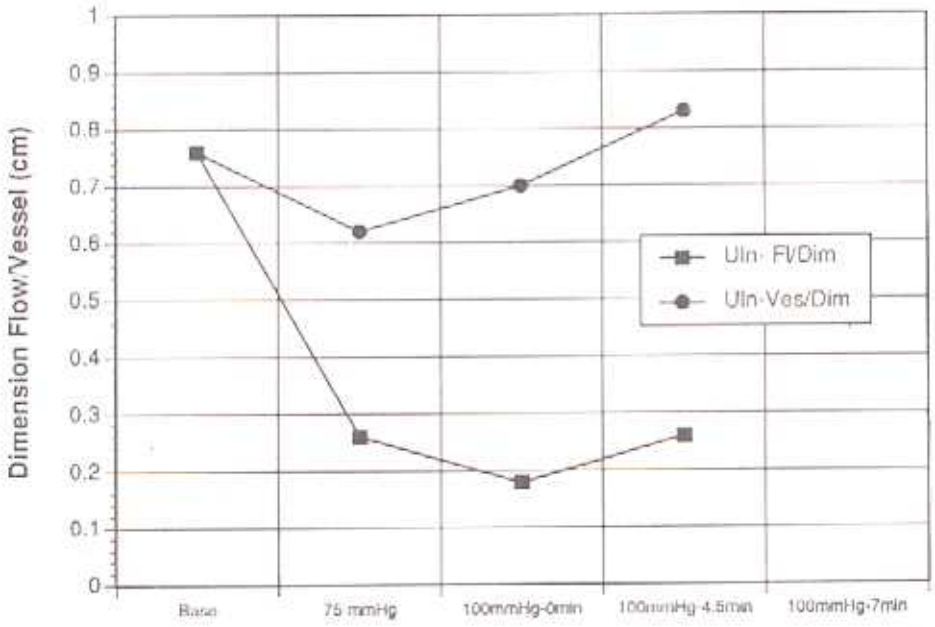


Figure 4. Blood flow and vessel dimension for ulnar artery.

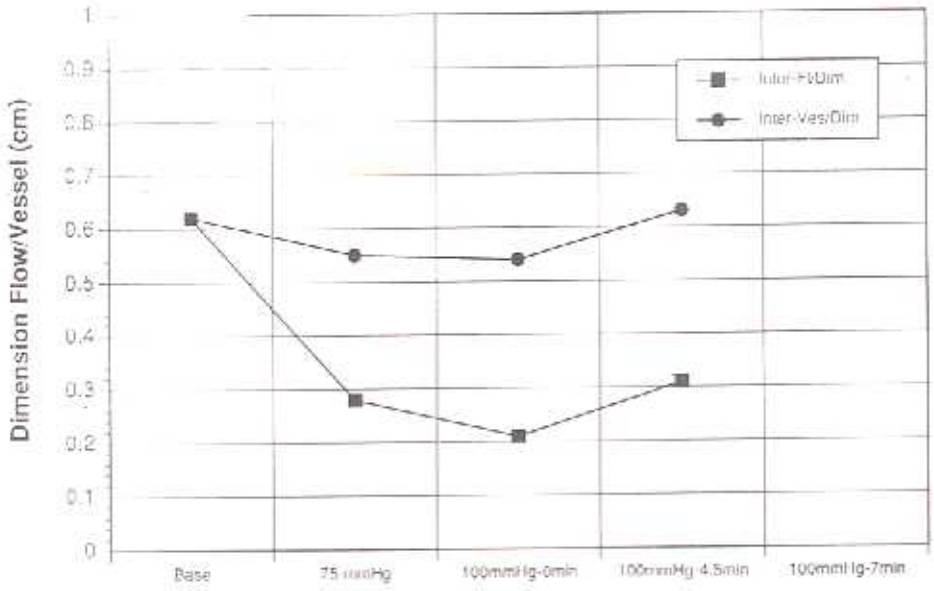


Figure 5. Blood flow and vessel dimension for interosseus artery.

congestion and ultimately arm pain will result – a condition of pain that is similar to that experienced during sustained G exposure. Sealing the arm in the chamber door is accomplished by an adjustable 'camera iris' opening that does not interfere with blood flow. The pressure in the chamber was maintained with anaeroid manometers with an error of less than ±5 mmHg.

Changes in regional volumes of the arm were determined by tetrapolar impedance plethysmography on the upper arm, where changes in regional blood volumes were estimated by observing the changes in resistance to a small-amplitude 50 kHz alternating current. Blood vessel size and blood flow were measured by sonography (Hewlett Packard SONOS 1500 Imaging System with an Acoustic Quantification 7.0 MHz sector high-frequency transducer). Finger blood pressure was determined by Finapres® (Ohmeda model 2300, Madison, WI). The arm pain was estimated by a subjective 0–11 unit scale where 0 indicates no pain at all, 5 indicates strong pain, and 11 maximal pain.

Results

Preliminary results indicate that with a 100 mmHg pressure increase in the chamber, arm pain occurred after 5–10 min. With higher pressure (125–150 mmHg) arm pain was greater and developed earlier. Data from one subject showed that the ratio between blood flow and vessel dimension in the radial, ulnar and interosseos arteries had decreased to less than half the control value after the chamber pressure was increased to 75 mmHg as indicated by sonography (Figure 2). The blood flow within the radial, ulnar and interosseos arteries decreased to about 1/3rd of the control value during 5 min at 75 mmHg hyperbaric chamber pressure and was maintained at about the same level during 4.5 min at 100 mmHg (Figures 3–5). The arterial sizes were maintained the same or with only minor changes during this exposure. The blood flow velocity decreased similarly to about one-half at 75 mmHg chamber pressure and decreased further to about 1/3rd in the beginning at 100 mmHg (Figure 6). After 4.5 min at 100 mmHg the

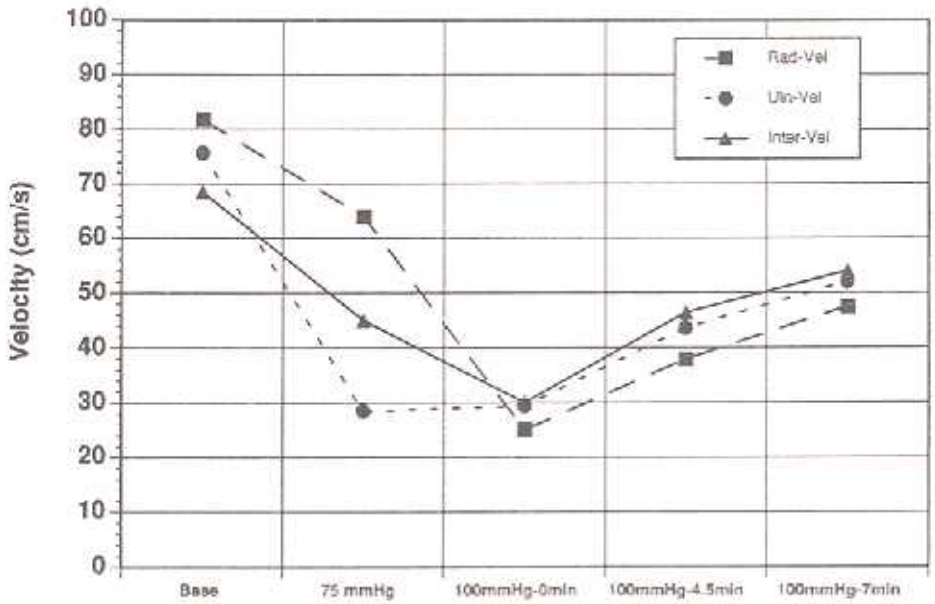


Figure 6. Blood flow velocity for radial, ulnar and interosseos arteries.

velocity returned somewhat and was about one-half of the control velocity after 7 min.

The arm volume increased very slightly with increased pressure in the pressure chamber in our preliminary measurements with impedance plethysmography. The arterial blood pressure in the finger rose approximately in proportion to the increased chamber pressure.

Conclusions

Arm pain, similar to that encountered during high G loads with PBG and/or a low-hand position, has been induced by exposing subjects to hyperbaric pressure in a chamber with the arm extended through an opening in the chamber exposed to atmospheric pressure. This differential pressure model simulates G-induced arm pain and provides a more convenient method to study physiologic changes. Also, countermeasures to reduce arm pain will be easier to test with this technique. Useful countermeasure techniques for high G protection must be validated using the centrifuge.

References

1. Burton RR, Leverett SD, Michaelson ED. Man at high sustained +Gz acceleration: A review. *Aerospace Med* 1974;45:1115–1136.
2. Prior AR1, Tozer A. Arm pain associated with positive pressure breathing for −Gz acceleration (Abstract). *Aviat Space Environ Med* 1992;63:400.
3. Prior AR1, Cresswell GI. Flight trial of an enhanced G-protection system in Hawk XX327. Farnborough, Hamsphire: RAF Institute of Aviation Medicine, 1989. Report No. 678.
4. Travis T, Morgan TR. US Air Force positive pressure breathing anti-G system (PBG): Subjective health effects and acceptance by pilots. *Aviat Space Environ Med* 1994;65 (5 Suppl):A75–79.