SAO Project Cover Page

Project 107
Project Title Human Spaceflight

Introduction

Immediately following the end of World War 2, the emergent world super-powers set about expatriating German scientists and physicians with a view to gaining the superior technology displayed by the V2 bomber the Germans had built. The USA's project Paperclip, the USSR's project Osoaviakhim and the UK's project Backfire, set these countries on a path of technological, political and social competition which would develop into the current international, intercultural space industry.

Space medicine was on the agenda. The acquired jet- and rocket-powered technology created new engineering challenges for protecting pilots. As a result of the nearly doubled speed of these new aircraft compared to anything already achieved, some immediately obvious concerns were highlighted. Ejecting from a cockpit at such speeds was likely to tear a pilot apart, reduced reaction times within the cockpit required a redesign, and noise management was important. Also, the greater altitudes exposed pilots to low air pressures and cosmic radiation, both requiring careful study and design and engineering considerations. The first university course in aviation medicine was begun in 1928 at Wurzburg, Germany after advice from Hubert Strughold, ex-Nazi physician, and subsequently known as the Father of Space Medicine (TSHAweb). Apparently, he was the first to coin the phrase 'space medicine' at a conference on Aeromedical Problems of Space Travel in 1948 (TSHAweb).

Harry Armstrong was instrumental in project Paperclip, and headed up the various incarnations of the US's School of Aviation Medicine (Mackowski 2006). A particular legacy of Armstrong's work is the Armstrong Limit, the altitude (around 12 miles) at which air pressure is so low that water boils at blood temperature, thus determining the limit of a pilot before a pressurized environment is needed (NASAExweb).

In 1949, to investigate high altitude radiation, the first animals in space were a canister of fruit flies which returned via parachute, from an altitude of 109 Km after ascending in the nose of Blossom 1, a modified V2 launched by the US Airforce Cambridge Research Centre in Ohio (Spacetodayweb, Reuter 2000). Later that year the ACRC launched the first mammal into space, an anaesthetised rhesus monkey called Albert (Reuter 2000).

In 1951, the USSR sent several dogs on sub-orbital trips to space, variously encased in suits and helmets, or in a pressurized cabin. Then, in 1957 the USSR put the first dog, named Laika into orbit, who unfortunately overheated and died within half a dozen hours of launch (Astronautixweb).

These were the fledgling attempts at discovering the effects of liftoff and weightlessness on mammals, crucial pre-cursors to sending humans into space. As we know, Yuri Gagarin made the first human trip to space, and to Earth orbit on 12th April, 1961. He wore an SK-1 full-pressure spacesuit, and life support and communications including video, were used to monitor his condition (NPOweb).

Humans have been in space for just 50 years, and space medicine is in its infancy. But we do have some data and some conclusions, thanks to the efforts and sacrifices of many. Human spaceflight has impacts on all the physical and psychological systems of the human body. Setting aside the huge field of psycho-social issues, such as were recently studied by the Mars500 project in Russia, and the issue of radiation exposure, this essay will look at some physiological consequences of the over-riding stressor of human spaceflight: gravity, the lack of it, and too much of it.

High Acceleration

Acceleration is the rate of change of velocity. Because deceleration is just negative acceleration, the latter term can be used to encompass the former. For our purposes, Newtonian laws of motion provide the underlying principles which explain the sensations experienced by a human subject undertaking a space craft launch or atmospheric re-entry. Newton's three laws are, briefly: a body in motion (or at rest) will continue in that motion unless acted upon by a force; acceleration is proportional to force and inversely proportional to mass; and a force directed by one body on another, is counteracted by an equal and opposite force.

In our space craft launch circumstance, a passenger at rest in her seat is rapidly subject to a force pushing her Earthwards as the space craft rises vertically from the launch pad, climbing out of the Earth's gravity well towards a velocity of 8 km/s, at which point it achieves orbit. During launch, she feels a force several times that of Earth-normal 1G which numerically is around 9.8 m/s/s. During orbit, our subject experiences weightlessness, effectively freefalling for the duration. Upon re-entry into the Earth's atmosphere, the space craft is subject to large deceleration forces as it encounters the drag provided by the increasingly denser environment. As the spacecraft rapidly loses velocity, the subject is forced against her seat harness, in the direction of flight.

Because humans have evolved physiologically in a 1G environment, then anything less than 1G is *relatively* negative, even if it's above zero G, and anything above 1G (9.8 m/s/s) is called *positive* G. The minimum amount of exposure to Earth-normal G for healthy human functioning is not known (Clement 2011), but the effects of positive G have been studied for a good 70 years.

Study into high speed acceleration and deceleration, amongst many other aspects of aerospace medicine, was first undertaken when Hitler poured large amounts of money into Germany's aviation research. By 1936, Germany had drawings for planes that could reach 500 mph, and cutting edge research on pilot ejection seats, and so pilot blackouts were going to be an issue. (Mackowski 2006) This section will be confined to the physiological effects of positive G as might be experienced during launch and re-entry of a space craft.

The first serious studies in human tolerance to high acceleration and deceleration were driven by John Paul Stapp at Muroc (now Edwards AFB) in California in the late 1940s. A rocket-propelled ejection seat was accelerated along a 2000 ft long track (the 'Gee Whiz track (Stappweb)) and brought to a rapid stop to study animal, then human responses to high 'G' loading. The oft-quoted truism "Murphy's Law" was born here when a technician connected every one of the 16 accelerometers on the harness the wrong way around, resulting in a dangerous test run which revealed no data at all. The technician's name was Edward Murphy. (Mackowski 2006)

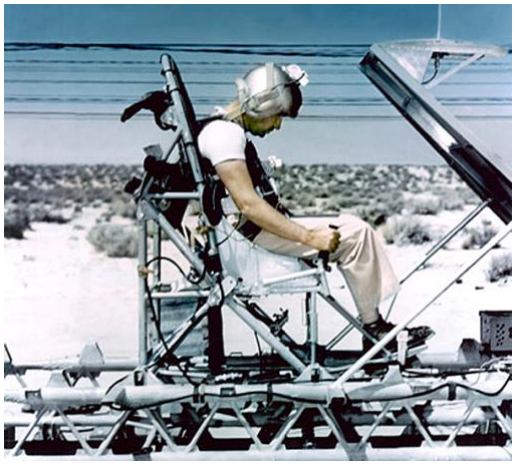

Fig. 1 The Gee Whiz rocket powered sled (Source: Fooyohweb)

Stapp maxed out at an instantaneous 46.2 Gs, and also achieved 25 Gs for over a second, crash survivors have experienced 100 Gs, Apollo re-entry subjected the occupants to as much as 7 Gs of deceleration (NASAJSCweb) and Mercury pilots up to 11G (Davis et al 2008). Director Stapp's Wright Laboratory in the US, tested the candidate Mercury astronauts (seated and lying down) in a centrifuge until each one became unconscious, revealing an average tolerance of 7 Gs – within specifications of von Braun's 9 G Mercury launch simulator, and a 12 G rocket trainer proposed by NACA (NASA's predecessor). At this stage, nothing was known of the Soviet research in this area, (Mackowski 2006). German and US researchers both discovered that the adverse effects of high G loading could best be ameliorated by orienting the subject at right angles to the acceleration, and specifically in Stapp's research, perpendicular to an imaginary line between the head and heart. Assessing the maximum G force sustainable by humans has been a high-risk endeavour, however, with a quarter of the USAF ejection seat tests up until 1955, being fatal (Mackowski 2006).

A distinction needs to be made here, between vertical and horizontal acceleration. Vertical acceleration is that which is directed parallel to the body's large blood vessels, that is, along the head-to-toe axis. Horizontal acceleration is that which is experienced perpendicular to vertical acceleration, that is, along the breast-to-back line. When standing on Earth, the human body absorbs the vertical acceleration of gravity through the larger bones of the lower half of the skeleton (lumbar spine, hips, thigh, calf, heal), and the larger muscles of the same region (rump, thigh, calf) (Clement 2011). When seated pre-launch in a space craft, the subject is positioned in an orientation with hips and knees bent at 90^{o} and above the chest, prepared for a horizontal acceleration. This minimises the effect of acceleration on blood movement through the long axis of the vessels, during launch or re-entry.

So what are the physiological effects of high G? During vertical acceleration, the body's thoracic and carotid pressure sensors signal the sympathetic nervous system to compensate for the lowering of blood pressure, by increasing heart rate, increasing heart stroke volume, and constricting muscles so as to reduce the volume of arteries. A lack of oxygen to the brain can cause cerebral hypoxia, but there's a lag time of around 5 seconds before brain function is impaired. The response of the sympathetic nervous system takes around 12 seconds, which is twice as long as the cerebral hypoxia reserve time, so brain damage is likely under high vertical G. This nerve reaction is automatically engaged by the 'fight or flight' response of the body's endocrine system, which becomes important in sustained high G environments. Vertical G also causes blood to pool at the bottom of the lungs where the alveolae are also shrunk or collapsed under differential pressure, oxygen absorption is decreased and lungs may collapse (Davis et al 2008)

Symptoms of exposure to high vertical G begin with tunnel vision, then loss of central vision (grey-out), then loss of vision altogether (black-out), and further exposure will lead to disorientation, amnesia, hearing loss, and eventually loss of consciousness. Experienced pilots can alert researchers to impending loss of consciousness by reporting their impaired visual symptoms (Davis et al 2008)

The hypoxia effects described above for vertical G, are very much reduced for horizontal G loads, because of the much shorter axis of effect across the main arteries of a reposed pilot. However, the difficulty of raising the chest to breath, the pooling of blood at the back of the lungs, as well as the shrunken alveolae, all result in a doubling of the effort required to breath at +4G compared to 1G, and a limit of about +15G for breathing at all (Davis et al 2008). NASA and Russia have adopted the horizontal position for launch and re-entry, although the shuttle astronauts return in a 'normal' seated position and experience 1.2G for 17 minutes. They also wear full pressure space suits with helmets, have a five-point harness, and firm, slightly padded and contoured seats to distribute loads across the body (Fig. 2) (Davis et al 2008).

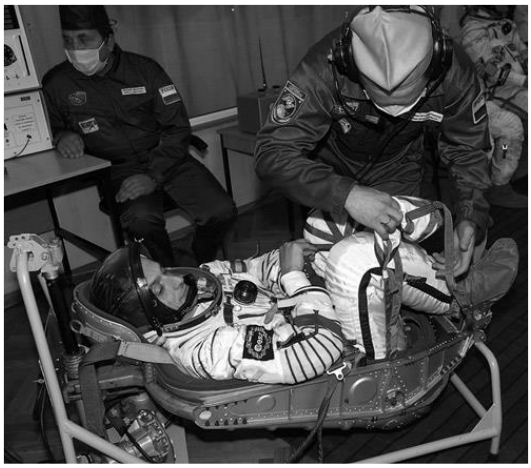

Fig. 2 The horizontal position assumed by astronauts for launch and re-entry (Credit: Clement 2011)

Space Sickness

Space sickness, space adaptation syndrome, space motion sickness. All labels for the contemporary motion sickness phenomenon experienced by space travellers. The first recorded episode of space sickness was from the second human to orbit Earth, Gherman Titov in 1961.

The word 'nausea' derives from the Greek word for 'ship' and reflects the roots of motion sickness in ancient seasickness. Pre-1900, the causes of motion sickness were variously considered to be related to imbalance in the vascular/intestinal systems, respiration problems, nervous shock, or some kind of infection. But, based on the work of Menier and others in the 19th century, the vestibular systems of the inner ear came under closer scrutiny. For a few decades after the second World War, it was thought that overstimulation of the semicircular canals was the culprit, but this was disproven by experiment. The well documented 'fluid shift' phenomenon in astronauts was popular for a while, but all these possible causes have given way to the current theory (Reschke 1990).

In the mid- to late 1950s, ex-Luftwaffe physician and pilot Harald von Beckh, had a water turtle with a damaged inner ear, which he used as a micro-g disorientation study subject in

parabolic aeroplane flights. The turtle rapidly out-performed other turtles which eventually learned to compensate for their apparent 'space sickness' and accurately targeted a piece of food held out for them (Mackowski 2006). Beckh's experiments laid the foundation for subsequent vestibular research in relation to space sickness.

The root cause of space sickness is not known, but the current best description is the sensory-conflict theory, proposed originally by Claremont in 1931 (Davis 2008). The inner ear vestibular system is strongly implicated here, because animals with their vestibule removed, and humans without such organs, are entirely unaffected by motion sickness (Davis et al 2008). Signals from the eyes, inner ears and receptors in the skin, muscles and joints, are mis-matched with expected outcomes of movement that the human brain is programmed to process. This causes symptoms such as lethargy, stomach upset, loss of appetite, headache, dizziness, drowsiness, pallor or flushing, nausea and vomiting (Clement 2011, Davis et al 2008, Hale 2010, Reschke 1990).

Even pilot trainers who are experienced flyers can be more susceptible to motion sickness in a training simulator than inexperienced students, because their brains receive slightly different inputs from the trainer, than they know from real flying. Characterised by sudden vomiting, space sickness is a form of motion sickness which generally increases in severity to a maximum within the first day, then drops off rapidly after the second day (Fig. 3) (Davis 2008).

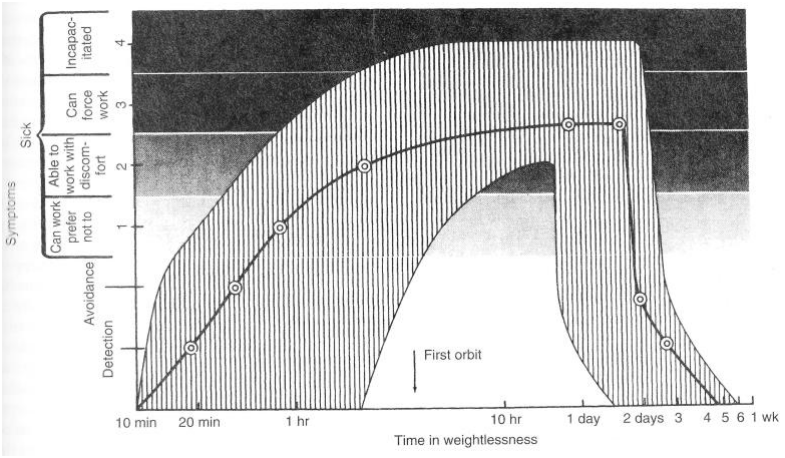

Fig. 3 Time of onset and progression of space sickness (Credit: Davis et al 2008)

Many people have experienced motion sickness in a car or boat or roller coaster ride, but an astronaut's susceptibility to space sickness cannot be extrapolated from their propensity to suffer terrestrial motion sickness. Probably 90% of the general population suffer a degree of motion sickness (Reschke 1990), but not all astronauts suffer from space sickness, some get a milder form (Sopite syndrome, a drowsiness possibly derived from the neonatal response of rocking a baby (Davis et al 2008)), and according to Clement (2011) there's no statistical difference between race, gender or experience, though Davis et al (2008) would disagree. The longer someone is on orbit, the more severe the symptoms and the longer the recovery (Clement 2011). Also, adapted space crew suffer (milder) symptoms for a few days after returning to Earth, but are less affected by other types of motion sickness during that time (Davis 2008). The only reliable symptomatology from Space Shuttle experiments and telemetry "…was that repeat flyers usually had fewer and less severe symptoms with each subsequent flight." (Hale 2010). Space sickness, though categorised as a motion sickness, appears to be not specifically caused by motion, and its epidemiology seems in dispute.

The sensory-conflict theory is supported by many observations. The rather confined Mercury and Gemini pilots reported no space sickness. One third of Apollo astronauts endured it, and as many as three quarters of STS astronauts and Soyuz cosmonauts reported some symptoms. This progression follows the increase in volume of the space crafts, and the amount of head movement involved (Clement 2011, Davis 2008). Refraining from moving their heads, particularly in the pitch and roll directions, and closing the eyes (Davis et al 2008), is a proven remedy, and symptoms often recede after a few hours. An interesting support of this theory came from the Apollo astronauts who walked on the Moon – they reported almost no space sickness before, during or after walking on the lunar surface (Homick & Miller 1975). The close association of the visual nerve input with the vestibular network, explains the impact that eyes have on motion sickness. A lack of 'grounding' reference points such as a horizon exacerbates the symptoms (Davis 2008).

It certainly seems that neither motion nor microgravity themselves are the cause, rather the perception of disorientation in a 'groundless' volume of space.

So, how does the vestibular system operate? Two separate, inner ear subsystems are at work here (Fig. 4). In the first, small Calcium Carbonate crystals called otoliths, are suspended in a gel in two chambers (the utricle and saccule), each of which have small hair cells lining the inner wall. When the head is subject to linear acceleration due to gravity, the otoliths move the hairs, which sends a signal to the brain interpreted as motion. The second system is not influenced by space flight (Clement 2011), and is a series of three semicircular canals filled with fluid which detect any *change* in motion.

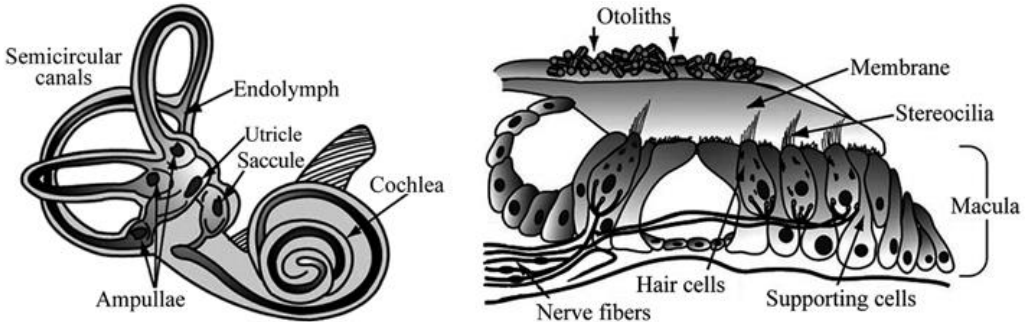

Fig. 4 Left: Fluid in the semicircular canals detects changes in motion. Right: $Ca(CO_3)_2$ granules called otoliths react to gravity and stimulate sensory hair cells. (Credit: Clement 2011)

Tilting the head forward by 30^o produces the same otolith response as experiencing 0.5g, so it is postulated that in microgravity, all signals from the otolith system are interpreted as resulting from linear head movement, and not from gravity (Parker et al 1985). This explains astronauts' staggering walk after landing (mal de debarquement), and feelings of body movement when they move their heads in space. But when further tested on STS mission 90, centrifuged astronauts reported feeling a tilt in their orientation, not a linear translation, a result contrary to this so-called Otolith Tilt-Translation Reinterpretation theory (Clement 2011).

There are other contributing factors to space sickness, though the extent and development of these are not well understood. Sensors in the skin, muscle and joints which normally tell us where our body parts are in space, send conflicting information to the brain when in microgravity. Either these proprioceptors have their function impaired, or the astronaut has an inaccurate perception of her environment (Watt 1997).

At the moment, management of the symptoms consists of working around the known timeline, and using drugs. Scheduled duties early on in the flight tend to involve less head movement, and EVAs are scheduled for no earlier than day 3 of a mission (Davis et al 2008). The risks associated with vomiting inside one's own helmet are obvious! Vestibular suppressants like scopolamine, Phenergan and promethazine (Davis et al 2008) can ameliorate symptoms while taking the drug, but may also impair the patient's ability to adapt naturally (Clement 2011).

This is an area of active research and some results from animals flown in space suggest that the amount of neural synapses in the hairs in the chambers which detect otolith movement, changes in response to varying exposure to gravity – that is, neural plasticity plays a role (Ross & Tomko 1998). There is clear evidence that space sickness is strongly related to a visual-vestibular mis-match in information supplied to the lower brainstem (particularly the limbic system and basal ganglia (Kohl 1987)), but the field is under close scientific questioning, with interest in improving pre-flight conditioning for space farers at the top of the list (Parker et al 1987).

Post flight Orthostatic Intolerance

Orthostatic intolerance literally means difficulty in standing upright, and is caused by three inter-related conditions exacerbated by space flight: reduced blood volume, reduced blood vessel ability to expand or contract, and heart function (Clement 2011). As alluded to in the section on high acceleration, the nervous system provides the impulses to secrete hormones like norepinephrine which manages the degree of blood vessel constriction, thereby controlling blood pressure in the tissues as the body moves relative to a gravitational load. The originating signals which determine the response come from baroreceptors, particularly in the carotid sinus, and it's not known if/how these receptors sensitivity change over long duration flights (Clement 2011).

Notwithstanding the ameliorating effects of a pressure suit, astronauts returning from a period of microgravity experience blood being pulled back down towards their legs. After having accommodated to low pressure in-flight for some time, the blood vessels are now subject to high physical stress, which causes them to dilate, allowing further blood flow into the legs. This results in blood flowing away from the head, and the potential for fainting (syncope). Because women have a higher heart rate and lower vascular resistance, they're more prone to these symptoms than men. And older people have a different response. When John Glenn travelled back into space on the space shuttle, at age 77, he recorded higher levels of norepinephrine than the other astronauts. This maintained his normal blood pressure before and after flight, by keeping vascular constriction higher (Clement 2011).

Specifically, orthostatic intolerance manifests as lightheadedness, high heart rate, low blood pressure, syncope, and decreased ability to exercise. The severity of symptoms is directly related to time in microgravity, as is the length of recuperation which is usually about a day for on-orbit durations less than a month, but up to several months for longer duration flights. Cosmonauts who've been on-orbit for six months or more are unable to egress from their return capsule without assistance.

Most astronauts suffer at least some symptoms, but very little data is available on the cardiovascular response of shuttle astronauts during re-entry. Also, because virtually no data exists for microgravity stays over nine months, some researchers fear that consequences such as reduced heart muscle mass, may render long-duration astronauts entirely unable to return to Earth.

When standing on Earth, blood in the human body is under the influence of gravity such that the pressure in the legs is around 200 mmHg, compared to around 70 mmHg in the head. This is countered by large muscles in the legs contracting and pushing the blood upwards to the heart, back-flow being prevented by one-way valves in the veins. Changes in posture trigger the release of norepinephrine in the appropriate locations, to constrict blood vessels and maintain correct blood pressure and supply. This response system appears to lose its functionality when someone has been in space for a while, where micro-G removes any hydrostatic gradients from the body. Carotid baroreceptors are implicated here, though little research has been done in this area.

Treatment for orthostatic intolerance is not governed by a full understanding of the problem. In-flight astronauts are required to drink plenty of fluids and take part in regular exercise, mostly to maintain cardio-pulmonary fitness, even though some studies suggest an increased incidence in aerobically fit subjects (Clement 2011). A Lower Body Negative Pressure 'suit' device has been used to apply a reduced pressure to the lower parts of an astronauts body, the body responding by increasing blood pressure and supply to the upper body, simulating the situation on Earth. There is however, considerable variation in results, and care must be taken to monitor heart rate and blood pressure.

An interesting approach to this problem, is for the astronaut to wear an elasticised 'loading' suit. The Russian Pingvin Suit (Fig. 5) applies two levels of vertical load to the body, by use of elasticised cords and a leather belt. This results in the wearer being forced to use their extensor muscles which subsequently pump blood back towards the heart.

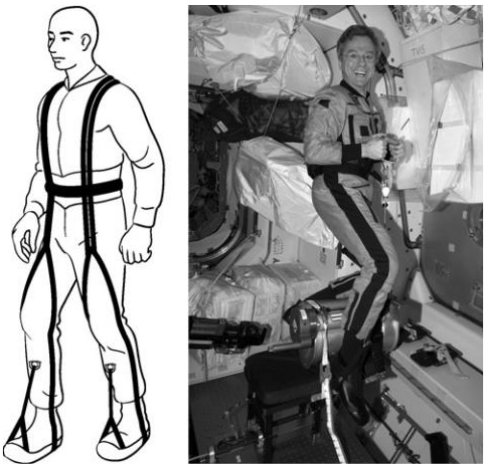

Fig. 5 The Russian Pingvin Suit assists venous blood pumping (Credit: Clement 2011)

A recent development in this technology saw the testing of a Gravity Loading Countermeasure Skinsuit (Waldie & Newman 2011), which is a skin-tight, elasticised garment providing hundreds of gradually increasing loads from shoulders to feet, simulating gravitational loads felt on Earth (Fig. 6). The garment can be adjusted to microgravity, or partial gravity as might be experienced on the Moon or Mars, and is comfortable enough to be worn as an undergarment and/or as a harness during training/conditioning exercises.

Though in-flight amelioration may help, and immediate post-flight treatment with the drug midrodine is very effective, orthostatic intolerance remains a poorly understood and studied condition, and its impact on future long duration flights is unknown.

Muscle Atrophy

Atrophy is the wasting away of tissue, and muscle atrophy in astronauts begins within days of exposure to microgravity, increasing with the length of stay in space. It may reach as much as 50% over longer periods, and is most obvious in those muscle groups associated with load-bearing postures on Earth. Without gravity, these larger back and leg muscles atrophy, whereas arm muscles tend not to suffer, perhaps partly because they're used more for translation around the spacecraft than the legs and back. (Clement 2011)

Metabolic processes like protein breakdown are responsible for the reduction in muscle fibre size, and their susceptibility to damage. This protein breakdown manifests as an increased excretion of nitrogen-containing species in the urine of in-flight astronauts. (Clement 2011)

Experimental investigation into muscle atrophy is hindered by several factors. Muscle biopsies are painful, and in-flight exercise, physical activity related to duties, and varying diets, all make it difficult to draw conclusive findings about loss of muscle bulk and strength. However, since oxygen consumption is somewhat related to exertion, then it can be used as an indicator of muscular activity. Combined with calf muscle biopsies of ISS astronauts, it seems as much as half the strength of both slow- and fast-reacting muscle fibres is lost over several months on orbit (Fitts et al 2004), the effect being only moderately slowed by exercise (Gallagher et al 2004), and recovery back on Earth being at least partly compensated for and masked by, the body's neuroplasticity and muscle protein supply (Riley et al 1996).

At the moment, it is not clear what biochemical changes are induced by microgravity, and therefore what actually causes muscle atrophy. This is a serious medical issue given the current excitement in many quarters about human colonization of the Moon and/or Mars. Work continues into cell physiology, endocrinology, exercise and pharmaceutical science in an attempt to address this issue.

Bone Loss

Bone loss mainly occurs in the spine and load-bearing leg bones, and occurs at around 2% per month in flight, long duration astronauts experiencing up to 20% loss, and the deterioration continues for months after return to Earth. Bone losses are greatest in the larger bones which are normally under highest G-loading, and is attributed to demineralization of calcium, the basic building block of bones tissue. Bone tissue formed in space is also weaker than normal. Modified bone marrow function also occurs, as well as increased risk of kidney stones due to the higher blood concentration of the released calcium. (Clement 2011)

Astronauts' bone loss via demineralisation of calcium (osteoporosis), approximates the condition studied in long bed rest patients on Earth, and although all patients lose bone tissue, there are no clear epidemiological trends. Even genetic predisposition appears to be a complicated process (Clement 2011).

Though the exact cellular mechanism for bone loss in not known, it seems that microgravity interferes with the formation of osteoblasts, the cells that create bone tissue. And this process is in part reliant on an enzyme called 'creatine kinase-B', study of which is current.

The risks to astronauts of bone loss, are increased propensity to develop osteoporosis and fractures post-flight (NASAHRRweb). As with muscle atrophy, an appropriate regime of load-bearing exercises, diet, therapy and pharmaceuticals is being developed to manage bone loss, but many unknowns remain.

Conclusion

NASA has in place a Human Research Roadmap (NASAHRRweb), which identifies some 31 specific risks to humans in space flight, ranging from radiation exposure to renal stone formation, orthostatic intolerance and psychiatric disorders. This essay has briefly touched on a handful of these issues which relate directly to the effects of gravity on the human body.

The variation in degree, and duration of exposure of humans to the force of gravity during the various stages of spaceflight, has an intertwining effect on many systems in the human body. Excessive G-loading during launch and re-entry can cause astronauts to blackout, but the adverse effects can be tolerated by appropriate physical orientation, seating and restraints. Space sickness is a largely vestibular phenomenon which can be managed with an appropriate activity regime design, and drugs. Orthostatic intolerance is a poorly understood consequence of the vascular system not coping with microgravity, and needs further study. It looms as a limiting factor for long duration space faring. Muscle atrophy and bone loss, though understood somewhat more in terms of mechanisms, lack a thorough physiological explanation, and also seem to pose a significant problem for the future of humans in space.

As a previously passionate proponent of human settlements on the Moon or Mars, it now appears clear to me that long durations in space are a long way in the future, simply because of the medical risks associated with a reduced gravity environment. Much more research is needed by way of physiological understanding, and management procedures.

References

Clement, G. 2011, Fundamentals of Space Medicine (New York: Springer, El Segundo: Microcosm)

Davis, J. R., Johnson, R., Stepanek, J., & Fogarty, J. A. eds. 2008, Fundamentals of Aerospace Medicine (Philadelphia: Lippincott Williams & Wilkins)

Fitts, R.H. et al 2004, TPh, 47, 320

Fooyohweb: Fooyoh web page, http://fooyoh.com/geekapolis_gadgets_camcorders/1604174 (accessed 6 Nov 2011)

Gallagher, P. et al 2004, TPh, 47, 321

Hale, W. ed. 2010, Wings in Orbit, Scientific and Engineering Legacies of the Space Shuttle, NASA/SP-2010-3409 (Washington: USGPO)

Homick, J. L. & Miller, E. F. II 1975, Apollo Flight Crew Vestibular Assessment, in Biomedical Results of Apollo, http://lsda.jsc.nasa.gov/books/apollo/S3CH8.htm (accessed 31 Oct 2011)

Kohl, R. L. 1987, AvSpEnvM, 58, 1130

Mackowski, M. P. 2006, Testing the Limits, Aviation Medicine and the Origins of Manned Space Flight (College Station: Texas A & M University Press)

McNamara, B. 2001, Into the Final Frontier, The Human Exploration of Space (Ft Worth: Harcourt College)

MSNweb, MSN web page, http://msnbc.msn.com/id/12869190/ (accessed 29 Oct 2011)

NASAExweb: NASA Explores web page, http://web.archive.org/web/20070927100528/http://www.nasaexplores.com/glossary_view_9-12.php (accessed 6 Nov 2011)

NASAHRRweb: NASA Human Research Roadmap web page, http://humanresearchroadmap.nasa.gov/evidence/ (accessed 29 Nov 2011)

NASAJSCweb: NASA JSC web page, http://lsda.jsc.nasa.gov/books/apollo/Resize-jpg/ts2c5-2.jpg (accessed 6 Nov 2011)

NPOweb: NPO International Cooperation In Space Zvezda Museum web page, http://www.npointercos.jp/Zvezda.html (accessed 29 Oct 2011)

Parker, D. E., Reschke, M. F. Arrott, A. P., Homick, J. L., & Lichtenberg, B. K. 1985, AvSpEnvM, 56, 601

Parker, D. E. et al 1987, AvSpEnvM, 58, A42

Reschke, M. F. 1990, Space Flight and Motion Sickness: A Historical Perspective, http://www.dsls.usra.edu/grandrounds/20080527/reschke.pdf (accessed 31 Oct 2011)

Reuter, C. 2000, The V2 and the German, Russian and American Rocket Program (Kanata: S.R. Research & Publishing)

Riley, D. A. et al 1996, JAPh, 81, 133

Ross, M. D., & Tomko, D. L. 1998, BrResRev, 28, 44

Spacetodayweb: Space Today web page, http://www.spacetoday.org/Astronauts/Animals/Dogs.html#OtherAnimals (accessed 29 Oct 2011)

Stappweb: Ejection Site web page, http://www.ejectionsite.com/stapp.htm (accessed 6 Nov 2011)

TSHAweb: Texas Stae Historical Association web page, http://www.tshaonline.org/handbook/online/articles/siarz (accessed 6 Nov 2011)

Waldie, J. M. & Newman, D. J. 2011, AcA, 68, 7-8, 722

Watt, D. G. D. 1997, AvSpEnvM, 68, 99

Wikiweb: Wikipedia Space Medicine web page, http://en.wikipedia.org/wiki/Space_medicine#cite_ref-1 (accessed 29 Oct 2011)