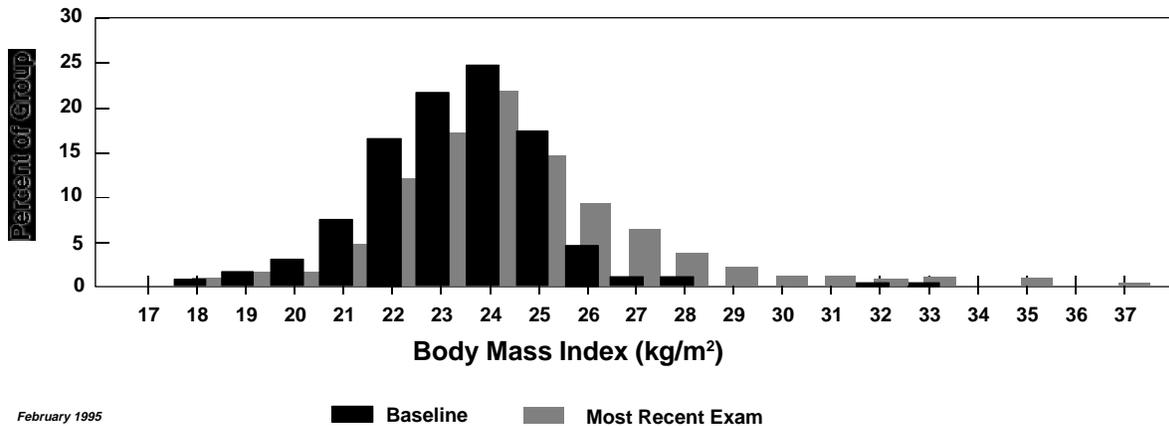


Study announcements continued from page 1

Update on Body Mass Index

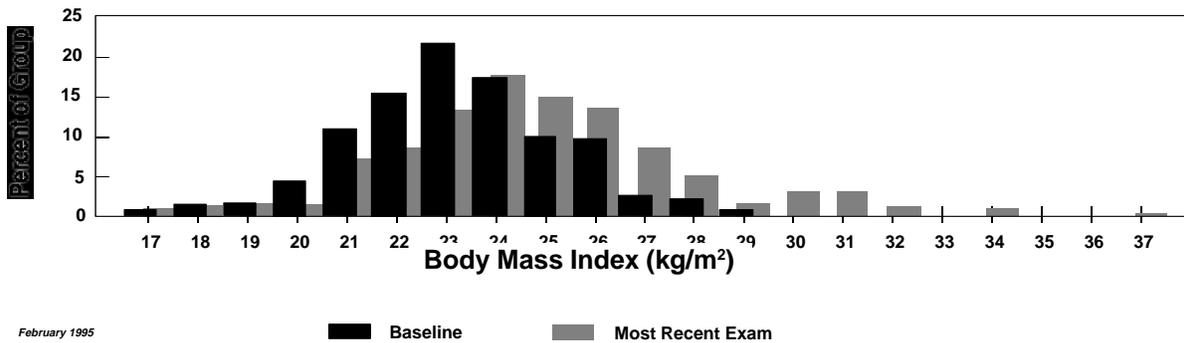
Body mass index is a measurement of weight adjusted for height, calculated as: $\text{weight (kg)}/\text{height (m)}^2$. A recent issue of the *LSAH Newsletter* reported body mass index measurements for LSAH participants at baseline. The mean values for the study group at baseline were 23.28 for the astronaut group and 23.34 for the comparison group. The most recent physical examination for each study participant has been reviewed, and the group results show a slight increase in body mass index because of weight gain as study participants age. For this assessment, weight and height for each study participant, as reported on the last physical examination completed in the JSC Clinics, were used. The mean body mass index for the most recent visit was 24.71 for the astronaut group and 24.37 for the comparison group (see the graphs below). The changes in weight over time for the comparison group is very similar to the change in weight for the astronaut group. Both groups show a small increase in mean weight since baseline.

Body Mass Index Distribution
 Baseline and Most Recent Exam
 LSAH Comparison Participants (N=658)



February 1995

Body Mass Index Distribution
 Baseline and Most Recent Exam
 Astronauts (N=219)



February 1995

Glaucoma: Are you at risk?

Glaucoma is one of the leading causes of blindness in the United States. A common threat to good vision in mid-life, it affects two out of every 100 people over the age of 40.

Glaucoma is a degenerative disease of the optic nerve. The optic nerve sends information from the eyes to the brain telling it what is being seen. It is made up of nerve fibers from all parts of the retina in much the way an electric cable contains a large number of wires. In glaucoma, some of the nerve fibers become damaged and fail to transmit signals to the brain. As a result, there may be blind spots which usually occur at the outer edges of the field of vision. This gradual loss of peripheral vision often goes unnoticed until significant optic nerve damage has occurred. If not treated, glaucoma may affect an increasing number of nerve fibers, causing continued shrinking of the visual field. The cause of the nerve damage in glaucoma is not known precisely. In most patients, it is associated with increased fluid pressure inside the eye known as intraocular pressure.

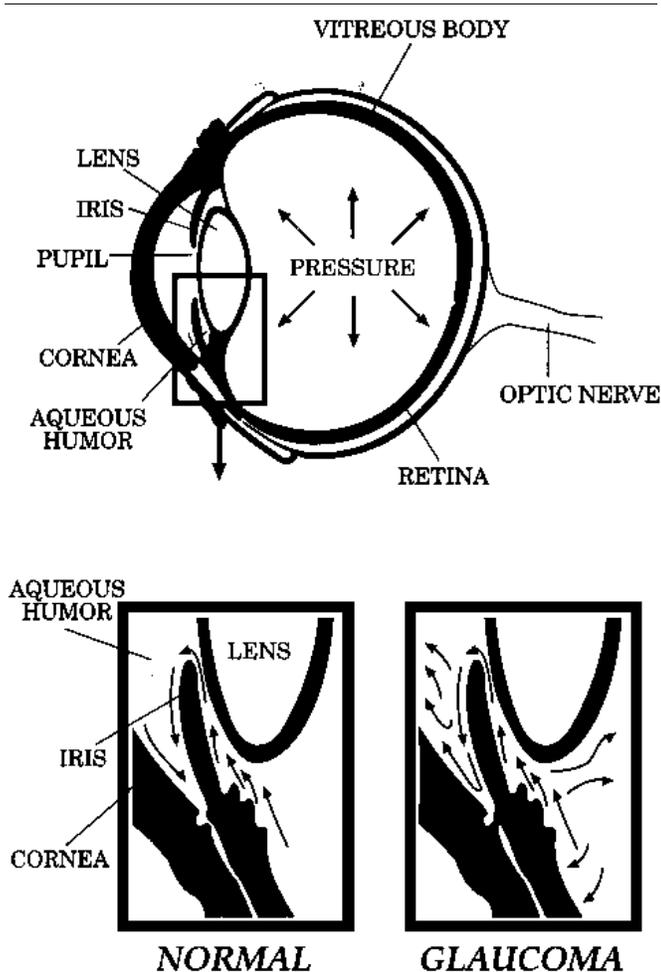
The eye constantly produces and drains fluid. If the drainage mechanism in the eye is blocked or restricted, the buildup of fluid will result in an increase in intraocular pressure. It is important, however, to understand that high intraocular pressure (i.e. ocular hyperten-

sion) does not necessarily result in glaucoma. There are individual tolerances for eye pressure and it can be higher than average in some individuals without resulting in

optic nerve damage or subsequent loss of vision. Most individuals with ocular hypertension go for years without developing nerve damage and may never have damage to the optic nerve. However, people with ocular hypertension have a higher risk of developing optic nerve damage.

Intraocular pressure is measured with a tonometer. Although several slightly different types of tonometers are used, the basic principle is the same. This test uses air pressure (in mmHg) against the outside of the eyeball to measure the resistant pressure inside the eyeball. This painless and non-invasive test is performed on both astronauts and comparison participants as part of the routine physical examination. Normal intraocular pressure is typically between 10 and 20 mmHg. If elevated intraocular pressure is detected, an in-depth evaluation of the eye is warranted before a diagnosis of glaucoma can be made. Other factors indicative of glaucoma include progressive changes in the optic nerve and corresponding visual field loss.

The optic nerve can be examined directly by looking at the inside of the eye with an ophthalmoscope whereas peripheral (side) vision testing is performed with an instrument called a perimeter.



The cornea and lens of the eye are bathed and nourished by a fluid (aqueous humor), which is constantly being formed and drained off. It circulates over the lens and behind the cornea before leaving through sieve-like outflow structures.

In glaucoma, the outflow structures fail to drain off sufficient fluid. Excessive pressure builds up within the eye. In time, the pressure caused by the fluid build-up destroys sensitive cells within the optic nerve and causes loss of sight.

Glaucoma continued from page 3

The graph below shows the intraocular pressures of the left eye, which was measured at the most recent physical examination for the astronaut population and the comparison population.

The right eye pressures were also examined and the distribution of values was very similar to the left eye. There was no significant difference in the distribution of intraocular pressures between the two populations. Mean values of the right eye were 13.5 mmHg for the astronauts and 13.2 mmHg for the comparisons.

There is no cure for glaucoma, but reduction of the eye pressure can slow or stop the progressive damage to the optic nerves. There are various treatment modalities (e.g., eye drops, oral medication, and surgery) available that are used to reduce pressure by improving fluid drainage. When glaucoma is recognized and treated early, loss of sight is almost always preventable.

Other conditions and characteristics that increase the risk of glaucoma include:

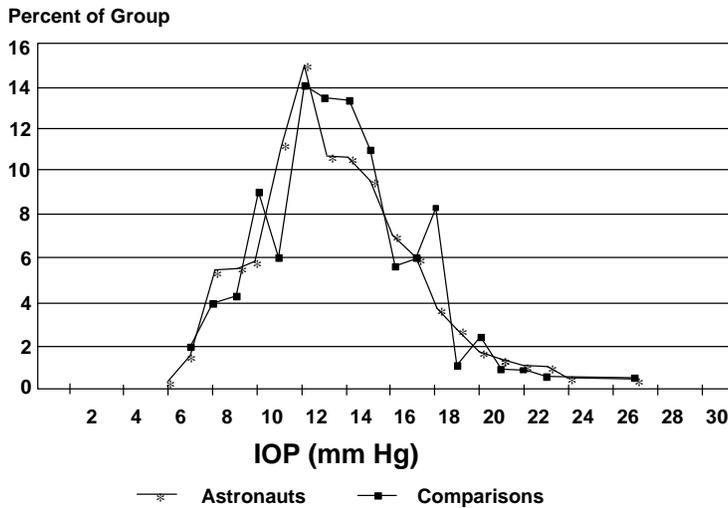
- aging,
- family history of glaucoma,
- African ancestry,
- diabetes,
- high blood pressure,
- long-term treatment with steroid medication, and
- eye injury or surgery.

It is possible to have glaucoma without experiencing any symptoms, but the common signs and symptoms include:

- frequent and unsatisfactory changes of eyeglasses,
- inability to adjust vision to darkened rooms, i.e., movie theaters,
- loss of vision,
- blurred or foggy vision, and
- rainbow-colored rings around lights.

If you experience any of these symptoms, discuss them with your doctor. An eye examination may be recommended.

**Intraocular Pressure (IOP) (mm Hg)
Measured at "Most Recent Exam"
Left Eye**



Mean values (mm Hg):
 Astronauts—13.7
 Comparisons—13.5

Serum CPK reported in LSAH participants

As with past newsletters, the LSAH continues to use the newsletter to inform you about various tests performed during your routine physical exams. This issue features the laboratory test for the enzyme Creatine Phosphokinase, or CPK.

CPK is an enzyme found in skeletal muscle, cardiac muscle, and the brain, all of which have elevated serum levels of CPK when stressed. Therefore, monitoring the serum level of CPK is important because elevated levels *may* indicate a serious health disorder, such as heart attack, stroke, or brain tumor.

However, other factors such as age, sex, and exercise also affect CPK levels. In fact, elevated CPK is common in adults who exercise regularly. Normal levels of serum CPK vary from 0 to approximately 188 micrograms per liter. Different laboratories may report slightly different upper limits as within the normal range because of differences in test procedures and population differences.

During exercise, especially of the high-intensity burst type, considerable amounts of CPK leak into the extracellular fluid and plasma as a result of changes in membrane activity. The type of exercise and the duration affect the elevation of serum CPK. Non-weight-bearing activities such as rowing and swimming cause less pronounced CPK elevations than comparable weight-bearing events. CPK elevations have been found to be linear with the duration of exercise up to 5.5 hours. Longer durations of exercise are associated with an accelerated increase in CPK.

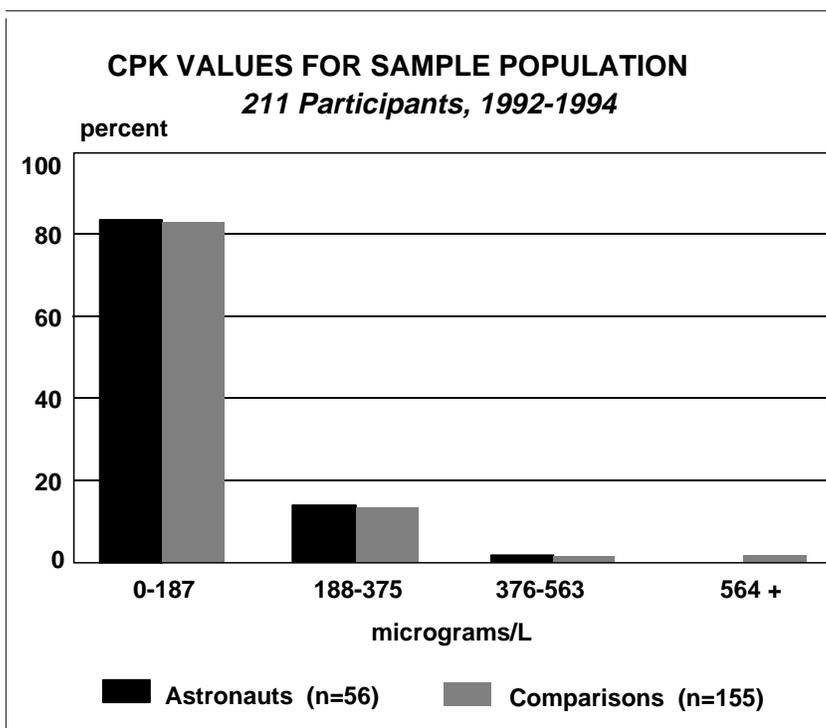
Exercise-induced increases in the level of serum CPK are temporary. Resting levels of CPK changes generally become elevated during exercise days and then return to normal levels during rest days. Peaks in serum CPK have been reported to occur at 5 minutes, 11 hours, 24 hours, 25 hours, and even 4-6 days following exercise. With continued physical activity, CPK levels remained elevated. Resting levels of serum CPK are higher in exercised trained individuals than in untrained ones.

CPK elevations vary with age and sex. Older individuals are less likely to have elevations after exercise than are middle-aged or young adults. Women are more likely to have exercise induced elevations than are men.

Some researchers suggest that muscle cell permeability increases during exercise and others think that muscle degeneration and necrosis occurs. Muscular soreness has been reported to accompany elevated CPK levels.

While increases in serum CPK are common in adults who exercise, there are also pathological states, such as myocardial infarction or heart attack, that may be accompanied by an increase in serum levels of CPK. Serum CPK

continued on page 6





CPK continued from page 5

is used as an indicator of muscle integrity with the level of specific isoenzymes aiding in the diagnoses regarding which type of muscle (cardiac, skeletal, or brain) has been stressed.

There are two isoenzymes, i.e., chemically different but functionally identical forms of CPK. The isoenzyme produced in the brain is identified as CPK B and the isoenzyme produced in muscle is identified as CPK M. As a rule of thumb, cardiac muscle stress produces CPK MB, and skeletal muscle stress produces CPK MM. CPK BB is an indicator of stress of brain tissue and is not usually examined in routine physical examinations unless there is a suspicion of stroke, tumor, etc.

There are divergent opinions about the proportion of CPK MB in skeletal muscle as well as about the proportion of total CPK in association with acute myocardial infarction. Studies have shown large elevations in CPK MB in some

women after step exercise and in highly trained female endurance runners. In the absence of symptoms and other positive physical examination information, elevated CPK MB is not usually considered sufficient evidence of a heart attack.

Identification of these isoenzymes requires laboratory testing procedures not routinely completed for LSAH. Approximately 16 percent of the 211 CPK tests completed for the LSAH participants during 1992-94 reported elevated serum levels (see graph). During this time period, about half of that 16 percent of laboratory tests were also examined for the isoenzymes. The isoenzymes identified in these tests have consistently been CPK MM. These data suggest that study participants are exercising prior to physical examinations. Questions regarding type and frequency of exercise routinely asked during the examination aid the physician in assessing the relationship between elevated CPK values and recent exercise.

If you want a copy of your exam results,

please complete and sign a release form while you are visiting the Clinic for your examination. The form is called *Privacy Act Disclosure Authorization and Accounting Record (DAAR)*, or NASA Form 1536.

If you have a new address or phone number,

please let us know by calling (713) 212-1362 or (713) 483-5785

or write us at

Longitudinal Study of Astronaut Health

Flight Medicine Clinic/SD26

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