$3,000 per week. None complained of respiratory symptoms on admission to the hospital or reported prior heart or lung conditions.

After administering several tests of pulmonary function, the investigators found that the subjects' total lung capacity was about normal. But 10 of the 19 people had a significantly decreased ability to incorporate fresh oxygen into their lungs and get rid of carbon monoxide; they also reported shortness of breath.

The cause of the defect in pulmonary gas exchange is uncertain, says Schnoll. It may be due to a contraction of the numerous blood vessels in the lungs after potent cocaine smoke is inhaled. Contaminants that are often added to cocaine sold on the streets may also harm lung function. Yet so far, he notes, the same effect has not been reported among cocaine snorters.

The researchers have evaluated nearly 120 additional cocaine smokers and found a similar loss of lung function among a similar percentage of them, says Schnoll.

"The observed lung damage may be short-lived," he adds, "but our tests show that it persists for at least three to five days after the cessation of freebase use." Two subjects examined 10 months after giving up cocaine smoking showed no improvement in their diminished pulmonary gas exchange. A larger population of cocaine users needs to be studied to sort out the drug's medical dangers, says Schnoll.

—B. Bower

Steroids heft heart risks in iron pumpers

The performance boost some athletes claim through the use of large doses of male sex hormones may be offset by an increased risk of heart disease, accumulating evidence indicates. One of the most recent studies, to be published in the July 27 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, documents the powerful effect that even four weeks of high-dose androgen use can have on transporter molecules in the blood, thought to protect against the clogging of vessels that supply the heart muscle.

Ben F. Hurley, an exercise physiologist who conducted the study at Washington University in St. Louis, monitored the levels of the transporter molecules, called high density lipoproteins (HDLs), in the blood of 16 healthy, highly conditioned men in their early 30s who were taking androgens in their training programs with heavy weights. Prior to steroid use, all the athletes had normal levels of HDLs (about 45 milligrams in 100 milliliters of blood). After four weeks of self-prescribed and -administered anabolic steroids, the levels dropped by about 60 percent.

Hurley, who is now at the University of Maryland in College Park, is cautious about interpreting the data. "You can't say that low HDL levels mean that a specific person is going to have a heart attack at an earlier age," Hurley told SCIENCE NEWS. "All you can say is that it increases their risk for heart disease." While his study and others indicate that the HDL levels return to normal within several weeks after androgen use stops, no one yet knows how an on-again-off-again cycle of hormone use in some athletes modulates the lipoprotein levels. "We have evidence that many of the athletes do not stay off the steroids long enough to get their HDL levels back to normal, therefore they place themselves at continuously high risk," Hurley says.

HDLs, tiny bags of linked protein and fat, are thought to act as garbage collectors in the human body's cholesterol-transport system, whereas LDLs, or low density lipoproteins, serve as delivery trucks to cells. Cholesterol is important in the construction of cell membranes, and as starting material in the production of many hormones. But when the LDL delivery trucks serve up more cholesterol than the HDLs can handle, the excess is sometimes dumped along the gutters of blood vessel highways to the heart. The result: gradually narrowing highways that can lay the groundwork for a heart attack.

The fundamental mechanism of cholesterol transport and its link to disease is still being worked out (SN: 4/22/78, p. 244), but "strong circumstantial evidence," based on epidemiological and biochemical data, indicate that high HDL levels should be protected, says John C. LaRosa, director of the Lipid Research Clinic at George Washington University in Washington, D.C. "I would not want to take a medication knowing that it lowered HDL levels unless there was a very good reason for it, and I would not think that [improved] body building or weight lifting would be a good reason," he says.

Hurley says the self-prescribed use of anabolic steroids among athletes who hope to better their performance seems to be increasing, although an increased risk of heart disease is only one of several health hazards associated with such use. Damage to the liver, kidney and reproductive system has been documented in previous clinical studies.

But do the drugs actually increase athletic performance? The American College of Sports Medicine (ACSM) recently revised its stand on the topic. While strongly discouraging use of the hormones among athletes, the ACSM admits that "gains in muscular strength ... can occur by the increased use of anabolic-androgenic steroids in some individuals."

—D. Franklin

Sperm antibodies frustrate fertility

Some infertile men appear to suffer from a confused immune system — they make antibodies against their own sperm. Researchers studying this disorder have purified a human sperm protein that could lead to the development of a rapid diagnostic test for some types of "immune infertility." The protein, which seems to play a crucial role in fertilization, may also have potential as a contraceptive vaccine.

Fertilization antigen (FA-1) was harvested from sperm cell membranes by a monoclonal antibody that binds it but ignores the sperm's numerous other surface proteins, according to a report in the July 20 SCIENCE by Rajesh K. Naz at the Oregon Regional Primate Research Center in Beaverton and colleagues.

To pinpoint the role FA-1 plays in fertility, the researchers coated human sperm with an antibody that locks onto this protein and then let the sperm loose on hamster eggs. Unlike normal human sperm, the coated sperm were unable to penetrate or even bind with the eggs. Fertilization of mouse eggs by mouse sperm was also inhibited by the antibody.

In a test that's closer to home—and that suggests a role for FA-1 in diagnosis of some types of immune infertility — Naz checked 15 infertile patients for antibodies against the sperm protein. Two of them, he found, had antibodies that "reacted very strongly" to the protein and five others had positive, but less strong, reactions, implicating the antibodies in some cases of infertility, Naz says.

Currently, Naz is collaborating with Allen Menge and his colleagues at the University of Michigan in Ann Arbor on contraceptive vaccine work. Menge reported last February that, using a similar technique, he'd purified a different sperm protein. Sperm treated with antibodies to this protein can fertilize eggs, but most of these eggs fail to implant in the uterus. Naz has vaccinated mice and rabbits with this antigen to induce them to make sperm-attacking antibodies and says he has "very good success" suppressing fertility this way.

Meanwhile, Erwin Goldberg of Northwestern University in Evanston, Ill., who purified the sperm enzyme lactate dehydrogenase (LDH-C4) over a decade ago (SN: 2/10/73, p. 95) and showed that antibodies against it reduce fertility in animals, says he is "encouraged" by his most recent experiments with a potential synthetic vaccine. Goldberg's group has synthesized parts of LDH-C4 and is testing them in female baboons. The only practical approach to a contraceptive vaccine, "both from the standpoint of quality control and also availability," Goldberg believes, is to use a synthetic antigen.

—G. Morse