Motion sickness is partly in the brain

The National Aeronautics and Space Administration can wrap astronauts in Gortex, place them in the most modern spacecraft, and propel them past the confines of the earth's gravity, but it can't free them from motion sickness. While NASA scientists have not solved the motion sickness mystery, they now suggest that a chemical in the brain is involved in the malady that causes such misery to travelers in space and on the ground.

Researchers Nancy Daunton of NASA Ames Research Center in Moffett Field, Calif., and George Crampton and James Lucot of Wright State University in Dayton, Ohio, report that a brain chemical secreted into the cerebrospinal fluid — the fluid that bathes the brain and spinal cord — causes vomiting during motion sickness. The scientists believe that motion sickness is caused when the vestibular system — the sensitive structures in the inner ear that are important in maintaining balance — sends abnormal signals to the central nervous system. These signals, and responses to a moving milieu by muscle receptors and vision, may combine to trigger the release of the brain chemical.

In animal studies, the researchers found that when they blocked the flow of the cerebrospinal fluid, motion-induced vomiting stopped. When the fluid was blocked only partially, vomiting was not suppressed. The precise chemical (or chemicals) has not been identified. Daunton says she suspects that it is always present, but that secretion increases during times of motion sickness. The chemical appears to trigger the vomiting, Crampton says, but it does not seem to cause the earlier, less extreme symptoms of motion sickness.

About 50 percent of astronauts and cosmonauts have experienced motion sickness during the first few days of weightlessness. Millions of travelers on earth also recognize the dreaded symptoms, such as disorientation, nausea and vomiting.

Daunton says that once the basic mechanics of motion sickness are understood it may be possible to develop an anti-motion sickness drug that responds specifically to the chemical. Achievement of this goal, however, depends on the chemical involved. "It may be something so ubiquitous that the blocking action may cause as many side effects as the drugs that currently are available," she says. Drugs such as Dramamine and Scopolamine are sometimes successful in preventing motion sickness, but they are "fairly nonspecific," she says, and no one knows exactly how they work. —*C. Simon*

PKU: Prenatal and carrier detection

Genetic engineering, the embryonic science of genetic manipulation, continues to revolutionize our understanding of the human genome and our clinical ability to treat defects therein. Weekly, cell biologists report new pieces to our chromosomal puzzle such as the recent location of a genetic marker for Huntington's disease (SN: 11/12/83, p. 311). Savio L.C. Woo and others at the Howard Hughes Medical Institute in Houston, Tex., report in the Nov. 10 NATURE another genetic giant step—the prenatal diagnosis and detection of phenylketonuria (PKU) carriers.

In PKU, the liver enzyme phenylalanine hydroxylase is absent, a condition that can result in severe mental retardation due to the buildup of phenylalanine and other substances in the blood. It is estimated that one in every 200 Caucasians is a carrier of the disease. PKU is transmitted as an autosomal recessive trait, meaning that when both parents are carriers each of their children has a 25 percent chance of having the disease and a 50 percent chance of carrying it. Until now prenatal diagnosis of PKU was not possible, nor was carrier detection, but infants could be tested for blood levels of phenylalanine and successfully treated nutritionally. Woo and co-workers report they are now able to detect carriers and affected fetuses of families with PKU history.

The technique used by the Baylor team is called restriction fragment length polymorphism (SN: 8/6/83, p. 90), where complementary DNA — one strand of the double helix—is used as a probe to detect individual discrepancies — polymorphisms—among parents and children in PKU families. Using restriction enzymes, which specialize in cleaving DNA between specific nucleotides, the researchers compared resulting fragments and found three that exhibited polymorphisms. Classical Mendelian analysis then made it possible for the researchers to sort out the fragment mixtures that showed up in affected children.

This test presently can detect prenatal defects for 75 percent of those parents who have had other PKU children and are concerned about future ones. The other 25 percent of existing PKU families are undetectable with the existing DNA probes, but more are being developed and Woo predicts that percentage will decrease rapidly in the future. The test may also benefit siblings of PKU children who wish to know if they are carriers themselves and people who are found to be carriers and wish to know if a developing fetus has the disease. As for the general population, the exact genetic mutations that cause PKU are not yet known and general screening procedures will not be available until they are. But the genetic revolution continues.

— *M. Wolfe*

Scientists give nod to sleeping pills

People suffering from brief bouts of insomnia — the kinds that result from bereavement or job stress, for example — should not be reluctant to take sleeping pills to help them through a few sleepless nights, according to scientists convened by the government to weigh the risks and benefits of "hypnotic" drugs. With the recent development of safer sleeping pills, physicians should no longer resist the idea of drug therapy for transient sleep disorders, the panel reported.

According to John S. Derryberry, a physician in private practice in Shelbyville, Tenn., and co-chairman of the panel, there has been a 50 percent decline in sleeping pill prescriptions over the past decade. While in general this may be viewed as a positive trend, Derryberry told reporters last week, it probably has resulted from patients' unwarranted fears of dependence and addiction; and it may have reached the point where sleeping pills are not being prescribed even when most appropriate. Specifically, the panel recommended as the "treatment of choice" a new class of benzodiazepines that are rapidly cleared from the body and, as a result, are less likely to cause the drug "hangover" associated with barbiturates and other traditional hypnotics.

For treatment of long-lasting insomnia, the panel was more cautious in its recommendations for drug therapy. The "insomniac" is a diminishing concept in medicine, the panelists concluded: When sleeplessness persists, it is almost always a symptom of an underlying disorder — either a psychiatric disorder such as depression or a medical disorder, such as sleep apnea. Treatment of the primary disorder usually leads to improvement in sleep, the report notes, but in the absence of certain medical conditions — alcohol dependence or apnea, for example — sleeping pills could be prescribed for "intermittent" use in combination with psychological and behavioral therapies. Only patients who fail to respond to such treatment over several months should be referred to a specialized sleep disorders center, the report says; such centers have become increasingly common in recent years, but their value has been a matter of considerable debate.

The panel, convened by the National Institutes of Health, reported some surprising statistics: In a national survey, one-third of the people reported some insomnia, and about half of those (or 17 percent of the population) considered their insomnia severe. But only 10 percent of those with severe insomnia were taking prescribed medication; the bulk of the prescribed hypnotics were going to elderly women with medical or psychiatric disorders. —*W. Herbert*