fied by their head-tossing, odd circling behavior, or other abnormal movements that seem to reflect balance difficulties stemming from inner ear problems.

Several years ago, Steel and her colleagues discovered that a mouse strain called *shaker-1* owes its hearing loss to mutations in the gene for myosin VIIa. This protein and many similar ones can bind to actin filaments and, through the effort of a built-in motor, move along them, often ferrying some form of molecular cargo.

Steel and her colleagues then realized that the human version of the gene for myosin VIIa resides in a part of chromosome 11 already implicated in one form of Usher's syndrome. Further research by Petit and other investigators established that mutations in this gene indeed cause the syndrome's visual and auditory problems.

Several cell types in the eye produce myosin VIIa, and according to studies conducted by Tama Hasson of Yale University School of Medicine, the protein is abundant in stereocilia and other specific regions of hair cells. "We know where the protein is, so now the question is, what does the protein do," says Hasson.

While the search for the protein's natural roles continues, Steel and her colleagues report in the November 1997 NEURON that myosin VIIa appears to play a role in the accumulation of a certain antibiotic inside hair cells. Consequently, the presence of myosin VIIa in the inner ear may explain why antibiotic-induced deafness is one of the major nongenetic causes of hearing loss.

As investigators continue to catalog new deafness genes, they need to firm up their estimates of how many cases of hearing loss can be attributed to each gene. At the moment, most of the excitement centers around connexin 26. The most dramatic evidence concerning its gene comes from a study by Petit and her colleagues in the November 1997 HUMAN MOLECULAR GENETICS. Among 65 families with histories of hearing loss, most of them from Tunisia, France, New Zealand, and the United Kingdom, the researchers found that about half had mutations in the gene for connexin 26.

"It looks like connexin 26 accounts for a lot [of deafness] in certain areas of the world, but it's not clear how much it accounts for hereditary deafness in the United States," says Friedman.

As the genetic data accumulate, physicians may begin to screen for mutations that might cause late-onset hearing loss or counsel people about potential outcomes of future pregnancies.

The latter issue is particularly important, since only half of congenital hearing loss stems from inherited mutations. "The main question of a family with a deaf child is whether they will have another one," notes Petit.

Outside the clinic, scientists will continue to examine how the proteins encoded by these deafness genes make hearing possible. They will investigate primarily the mouse inner ear, especially in mice bred to have mutant copies of the various deafness genes.

Mice will also serve as the initial testing ground for any treatments that might emerge from the discovery of deafness genes. Last year, for example, Anil K. Lalwani of the University of California, San Francisco reported at a meeting that he and his colleagues had for the first time successfully introduced a foreign gene into the mouse cochlea. With the growing number of identified deafness genes, physicians should soon know which ones will be useful in gene therapy for preventing or reversing hearing loss. □

Behavior

To dream, perchance to scan

Brain scans obtained from sleeping men have helped illuminate the neural system that makes dreaming possible, reports a team of investigators.

During rapid eye movement (REM) sleep, the brain's visual system retains its power to generate images but cannot process external sensations, the group notes in the Jan. 2 SCIENCE. Several inner-brain structures involved in memory and emotion exhibit heightened activity during REM sleep. In contrast, frontal lobe areas that integrate visual information with other sensations and make possible temporary recall of related items display sharp drops in neural effort, contends neuroscientist and report coauthor Allen R. Braun of the National Institutes of Health in Bethesda, Md.

These physiological features of REM sleep lay the groundwork for characteristic elements of dreams, such as intense emotions, bizarre happenings that seem unquestionably real, fractured time sequences, and a surreal lack of reflection about all the strange goings-on, the researchers theorize.

Braun and his colleagues obtained positron emission tomography (PET) scans of blood flow in the brains of 10 men during REM sleep and compared the images to scans taken during another stage of sleep—slow wave sleep—and to scans taken while the men were awake. Dreams occur most often and most vividly during REM sleep, the investigators assert.

REM sleep was characterized by low blood flow in the frontal lobes and in the tissue at the back of the brain that receives initial visual information from the eyes. A drop in flow reflects lessened neural activity. Blood flow surged in areas that orchestrate visual scenes and that contribute to emotion and long-term memory.

In an earlier PET scan study, researchers had found that the amygdala—a brain structure involved in processing fear and other emotions—becomes particularly active during REM sleep (SN: 9/21/96, p. 184).

Long-time sleep researcher David Foulkes, formerly of the Georgia Mental Health Institute and now working independently in Atlanta, doubts that these new brain studies shed light on the anatomy of dreaming—precisely because they focus solely on REM sleep.

Much evidence suggests that dreams of the same kind and of comparable intensity occur in nonREM sleep, Foulkes argues. Thus, he says, no physiological property unique to REM sleep can explain dreaming.

In the absence of environmental input or voluntary self-control, consciousness—a cognitive system for producing awareness of sensations and memories—creates dreams, Foulkes theorizes. Under some circumstances, ranging from relaxed wakefulness to REM sleep, this mechanism results in dreaming.

People develop the capacity to dream during childhood, says Foulkes, who notes that dreams do not assume a story-like form until age 7 or 8. Maturation of the frontal lobes may be required for dreams with a narrative structure, in his view.

The new PET findings offer valuable insights into the anatomy of dreaming, counters psychiatrist J. Allan Hobson of Harvard Medical School in Boston. Optimal brain conditions for dreaming exist only during REM sleep, he holds.

Braun's data support the notion that dreams reflect the activation of an individual's "emotional memory bank," with little direction or interpretation from the frontal lobe, he proposes.

"The PET results are consistent with Freud's idea that dreams have meaning," Hobson says, because they draw on neural repositories of memories. "But they challenge his theory that an unconscious dream censor [which would be located in the frontal lobes] screens out and masks particularly disturbing wishes."

Whatever the anatomy of dreaming, notes Braun, the core functions of REM sleep remain poorly understood. —*B.B.*