Biology

Julie Ann Miller reports from the meeting in Dallas of the Society for Neuroscience

Ever-changing shapes of nerve cells

The complex treelike silhouette of a mature nerve cell can vary continuously, with individual branches extending, retracting, disappearing or forming anew, report scientists at Washington University Medical School in St. Louis. Many of the signals from other nerve cells are received along these branches, which are called dendrites. Long-term changes in the nervous system occur at specialized communication sites, known as synapses. An important question has been whether the long-term changes involve primarily alterations in the function of existing synapses – for example, those changes described in the marine snail *Aplysia* (SN: 11/16/85, p. 308) – or whether there are structural changes in the nerve cell.

A new technique now allows scientists to analyze the shape of an individual nerve cell over days and months. Robert D. Hadley and Dale Purves have examined mouse nerve cells on the surface of a cluster of cells, called the superior cervical ganglion, located in the animal's neck. They anesthetize the mouse and photographically record the location of a nerve cell. Then they inject the cell with a nontoxic fluorescent dye that diffuses into the dendrites, revealing their shape and length. The animal is allowed to recover from the anesthesia. A few days to a few months later, the procedure is repeated to provide a second view of the same cell.

"Dendritic arbors contract and extend, but mostly extend," says Hadley. He reports an average dendritic growth in young adult mice of about 10 percent during three to seven days. After longer periods, there were progressively greater changes of dendritic geometry. "The subtle changes in single cells that we describe would have been impossible to discern by looking at populations of neurons with conventional means," Hadley and Purves say. "These morphological changes are almost certainly associated with functional changes in the synaptic circuitry." They conclude, "Such modulations of connectivity may bear on the cellular basis of long-term change in the central nervous system."

Two genes for Joseph disease

Two genes, located on different chromosomes, are associated with Joseph disease, a fatal genetic disorder in which loss of specific brain cells leads to paralysis. The first gene, found on human chromosome 1, appears to cause the disease, which is inherited as a dominant disorder. A second gene, called the modifier gene, reduces the severity of, or even eliminates, Joseph disease in persons who are expected to develop the disease. Abraham I. Grossman of the University of Texas Health Sciences Center at Dallas and his colleagues have located the modifier gene on human chromosome 2.

Listen to the ears

The human ear emits soft sounds both in the absence of external stimulation and in response to noise, scientists recently reported. While the biological value of this ear sound is unknown, it is expected to be a valuable tool for studying the workings of the inner ear. Brenda L. Lonsbury-Martin, Alfred C. Coats and their colleagues at Baylor College of Medicine in Houston now report a survey of monkey ears. The researchers sealed miniature microphones into the external ear canals of 41 macaques, 10 baboons and 10 squirrel monkeys. Only three monkeys, all macaques, displayed multiple spontaneous emissions. In contrast, in human studies investigators have detected such emissions in almost half their subjects. The bandwidth, frequency and level of the monkey ear emissions were similar to those reported for humans, so the low incidence is probably due not to differences in inner ear structures but rather to the mechanism that generates the ear sounds.

Biomedicine

Joanne Silberner reports from the American Heart Association Scientific Sessions in Washington, D.C.

The importance of having collateral

The body has a backup system for bringing blood to the heart muscle. And the extent of the backup system may determine an individual's likelihood of surviving a heart attack, according to Marc Cohen and Peter Rentrop of the Mt. Sinai School of Medicine in New York City.

The heart muscle is fed by coronary arteries; when a large one is blocked, the backup system – nearby collateral arteries – sometimes fills in. But often the process is not enough to keep a critical situation from becoming fatal. While people who have good collateral development following a heart attack are generally thought to have a better prognosis than those who don't, this is the first trial to show that some people have a better collateral system than others even before a heart attack occurs, Cohen says.

The collaterals aren't used by healthy hearts, so to get a picture of them Cohen and Rentrop took advantage of coronary angioplasty (SN: 11/29/80, p. 341). In the process, a tiny balloon is threaded into a clogged coronary artery and inflated, enlarging the bore of the vessel. Heart muscle downstream from the balloon is temporarily deprived of blood flow, creating a mini (and reversible) heart attack.

Cohen and Rentrop injected a radiopaque dye into the hearts of 21 patients getting coronary angioplasty and used X-rays to watch the dye backflow into the blood-starved area. They found differing degrees of filling, and in two patients no filling. Patients with a better collateral system had a smaller segment of the heart that stopped moving when the blood supply was down, and patients without collaterals had a larger segment that stopped moving, Cohen says. Why some people have better collaterals, and thus a better chance of surviving a heart attack, than others remains to be determined, he says.

The heart and heredity

Some of the complicated ways in which genes control cardiovascular function are beginning to come to light. According to a study from the University of Iowa in Iowa City, an inherited difference in blood flow patterns that may presage hypertension can be seen in childhood. And research at the Medical College of Virginia in Richmond indicates that there are two genetic factors controlling blood pressure and heart rate changes.

The Iowa study compared 13 children 12 to 18 years old whose parents had normal blood pressure with 12 children having at least one parent with hypertension, putting the children at higher risk of eventually developing the condition themselves. The children were put in a blood pressure-raising situation – instructed to mentally subtract a two-digit number from a four-digit number in a specified period of time.

"We've found evidence of a different pattern in children genetically susceptible to hypertension," says Erling A. Anderson of Iowa. The difference was not in blood pressure or heart rate, which remained essentially the same in the two groups both before and after the stress, but in a significant increase in blood flow to the forearm in the children of hypertensives.

The Medical College of Virginia study suggests there may be two genetic components controlling heart rate and blood pressure. Researchers administered exercise tests to 83 sets of identical twins and 57 sets of nonidentical twins. Before and after work, they found a tighter correlation for blood pressure and heart rate values in the identical twins than in the nonidentical twins, as would be expected for a trait under genetic control. But in analyzing the *rate* of increase from rest to mental or physical work they found no significant similarities in either group of twins. This, they say, suggests there are different genetic factors controlling the relatively quiescent heart and the heart at work.