BEHAVIOR

Emotional brain waves

It'll be a long time before anyone is able to tell what you're thinking by looking at printouts of your brain waves, but bit by bit the electrical activity of the brain is being interpreted. Electrodes are placed at various locations on the scalp and the brain's electrical activity is detected and recorded as an electroencephalogram. On the EEG there are subtle brain wave patterns that are now known to occur in response to specific stimuli. The P 300 wave, for instance, occurs in response to a sudden stimulus, such as an alarm or a flashing light. The wave is designated "P" because it is positive and "300" because it occurs 300 milliseconds after the stimulus. Researchers at the Downstate Medical Center of the State University of New York in Brooklyn are now studying a P 3 wave that occurs only in response to emotional stimuli.

Henri Begleiter and colleagues have found that the P 3 wave comes not from the cortex, where thought takes place, but from the lymbic system, where feelings arise, and in experiments it is seen only under specific conditions. When research subjects, for example, are asked to respond to different numbers (say, 0 and 1) by pushing specific buttons (A and B), no P 3 waves are seen in the printout. "But if you tell the subject you will pay a dollar for every correct push of button B," explains Begleiter, "what a difference." The P 3 wave occurs again and again.

The P 3 research may eventually prove useful in the early detection of certain brain disorders, suggest the researchers. Schizophrenics and alcoholics, for example, who show many signs of impaired emotional response, also show much lower P 3 waves than do normal subjects. "What this means, among other things," says Begleiter, "is that we now seem to have a way to show dysfunction even before it shows up as noticeable symptoms ... even when there is absolutely no physical damage showing up on a CAT scan."

The dream director

Did you ever have one of those dreams in which everything goes wrong—the kind in which you fall off a cliff or drown or get assaulted? Stephen P. LaBerge did, but he doesn't have to anymore. He says he can get inside his dreams and change the plots while they are still in progress. In the January PSYCHOLOGY TODAY LaBerge, now at Stanford University School of Medicine's Sleep Research Center, describes the phenomenon of "lucid dreaming" and explains how he taught himself to control it.

Lucid dreaming, or dreaming while being fully aware that you are dreaming, has been known of since at least the time of Aristotle. During such dreams, explains LaBerge, "the dreamer's consciousness seems remarkably wakeful. The lucid dreamer can reason carefully, remember freely and act volitionally ... the dreamer may take an active hand in resolving the dream's conflict and in bringing the plot to a satisfactory conclusion."

LaBerge has devised a method that allows him to produce lucid dreams "virtually at will." He calls the method MILD — mnemonic induction of lucid dreams. It is based on the formation of mental associations between what one wants to remember to do and the future circumstances in which one intends to act. The associations, LaBerge explains, are most readily formed by the mnemonic device of visualizing oneself doing what one intends to remember to do. It is also helpful, he says, to verbalize the intention. Before going to sleep, for instance, he says to himself, "Next time I'm dreaming, I want to remember I'm dreaming." Then he visualizes himself lying in bed dreaming, and at the same time he sees himself as being in the dream and realizing that he is in fact dreaming. The MILD method eventually enabled LaBerge to produce an average of 21.5 lucid dreams per month, with as many as four in one night.

ENVIRONMENT

Mutagens in diesel exhaust

Many of the 1,000 or so organic compounds in diesel exhaust cause mutations — genetic damage — in bacteria. A number of them have also been shown to cause cancer in mice when applied in concentrated form to their skin. But the carcinogenic compounds and nearly all identified mutagens wreak biological havoc only after activation by mammalian enzymes. Now, a team from the University of California at Berkeley reports what they believe to be the first "directly mutagenic" class of compounds found in diesel exhaust — compounds that inflict damage without requiring activation by body enzymes.

Writing in the December ENVIRONMENTAL SCIENCE AND TECHNOLOGY, Stephen M. Rappaport, Yi Y. Wang and colleagues suggest these "direct-acting mutagens in engine-exhaust particulates may represent a new and hitherto unrecognized class of environmental toxins." What's more, their work indicates "that virtually all of the mutagenicity of extracts of diesel-exhaust particulates was direct acting." It also appears, they say, that direct-acting mutagenicity "is based upon the collective action of several mutagens ... probably several distinct chemical classes of mutagens."

Finding PDAA (pyrene-3, 4-dicarboxylic acid anhydride) and probably other members of its chemical class in engine exhaust "is intriguing," the researchers say, because in theory they possess all the structural and chemical attributes needed to be among the direct-acting mutagens sought. PDAA is only weakly mutagenic. Other yet-unidentified chemical cousins, however, might prove considerably more potent.

. . . in animal tests

An animal test to examine diesel-exhaust effects at concentrations and exposure times occurring in the environment has just been reported by researchers at the Pasadena Foundation for Medical Research. Called "Sister Chromatid Exchange," the sensitive test involves placing suspensions of diesel-exhaust particles into the lungs of hamsters and then analyzing colonies of cells from those lungs that have been removed and grown outside the body.

For analysis of the cells, one chromosome in each chromosome pair is stained darker than the other. Under a microscope, exchange of genetic material between members of a pair is watched. The amount of exchange—which connotes mutagenicity—is dose related. Initial tests indicate most mutations occur in the first three months of exposure to exhaust particulates.

. . . and in the Big Apple

Direct-acting mutagens (those not requiring biological activation) have been identified in the air about New York City. But a seasonal variation in their concentration suggests that their predominant source is other than vehicle-exhaust fumes. In fact, burning fuel oil for space heating probably contributes half of the mutagenic activity per cubic meter of air in the Big Apple during winter, according to research by Joan M. Daisey, Theodore J. Kneip and colleagues of the Institute for Environmental Medicine at New York University.

The mutagenic activity of winter air is roughly twice that of summer. One reason is an increase in airborne-particulate concentrations. But space heating contributes as much mutagenic activity as all nonseasonal sources combined — including vehicular emissions. And the NYU scientists note that the particulates they measured seem more active per unit mass in winter than in summer. This has led to their provocative speculation that it may be that "organic materials emitted by space heating are inherently more mutagenic."