Panel urges treatment for panic disorder

Effective treatments, both psychological and pharmaceutical, exist for panic disorder, a condition that strikes about one in 75 people at some time in their lives. Unfortunately, no systematic studies exist to guide physicians and mental health clinicians to the best form of treatment for specific cases of panic disorder, concludes a report issued last week by a panel of psychiatrists and psychologists convened by the National Institutes of Health in Bethesda, Md.

Panic disorder involves recurring panic attacks, 10- to 15-minute episodes in which an overwhelming fear of imminent death, going insane or loss of control takes over. Physical symptoms such as shortness of breath, dizziness, racing heart, nausea and chest pain often accompany the attacks. Panic disorder sufferers often experience the episodes of terror at random moments, although they frequently occur in association with stressful events, such as surgery, pregnancy and heavy caffeine intake.

At least one in three panic disorder sufferers develops agoraphobia, a fear of places or situations that they feel might trigger a panic attack or, in the event of an attack, hinder their escape or the delivery of help. Severe agoraphobics rarely venture from their homes.

Some psychoactive medications ease panic symptoms, the NIH panel asserts. These include two classes of antidepressants – tricyclics and monoamine oxidase inhibitors – and three tranquilizers classed as benzodiazepines. Cognitive-behavioral therapy – designed to change mistaken beliefs about normal physiological reactions when anxious and provide gradual, supportive exposure to feared situations – also serves as an effective treatment, the panel adds.

"To some degree, these have been dueling therapies," says panel chairman Layton McCurdy, a psychiatrist at the Medical University of South Carolina in Charleston. Psychiatrists generally emphasize drug treatment for panic disorder and believe the repeated attacks stem from an imbalance of specific chemical messengers in the brain, McCurdy notes; psychologists stress cognitive-behavioral approaches and argue that panic disorder results from misinterpretations of bodily responses to normal anxiety.

As a result, panel members could find no studies comparing the two categories of panic disorder treatments or charting their combined use. The first such investigation, now underway at four universities and directed by psychologist David H. Barlow of the State University of New York at Albany, remains in its early stages.

However, a return of panic symptoms apparently occurs much less often after cognitive-behavioral therapy, compared with drug treatment, McCurdy remarks.

For now, the panel recommends that clinicians reassess using any treatment that fails to reduce panic symptoms within eight weeks.

Although the report takes a "balanced and judicious" stand, panic disorder still evokes considerable controversy, says psychiatrist Gerald L. Klerman of Cornell University Medical College in New York City. Klerman did not sit on the NIH panel, but he notes that debate centers on whether panic disorder represents a diagnosis distinct from more general forms of anxiety, concerns over the addictive potential of benzodiazepines and questions about the actual efficacy of psychological treatments. – *B. Bower*

UV pours through ozone hole

Even as this year's ozone hole opens over Antarctica, scientists report that the region received a dramatically high dose of ultraviolet radiation in late 1990 as a result of last year's ozone hole.

During December, the beginning of the austral summer, levels of damaging ultraviolet light last year registered twice their normal value, according to John E. Frederick and Amy D. Alberts of the University of Chicago, who made their measurements at Palmer Station, a U.S. base on the Antarctic Peninsula. The radiation reaching Antarctica last summer may have been the strongest the region has experienced since the ozone layer formed about a billion years ago, the researchers assert in the October GEOPHYSICAL RESEARCH LETTERS.

Ultraviolet light from the sun can harm humans, plants and animals. In Antarctica, biologists have found such radiation damages the DNA of certain species of phytoplankton, tiny floating plants that are a critical component of the polar food web. The seasonal hole began forming in the late 1970s because of increasing concentrations of ozone-destroying chlorine pollutants in the stratosphere.

Ultraviolet radiation reached such strong levels in 1990 because the springtime ozone depletion persisted longer than it had in past years, the researchers say. Winds from the north normally invade the Antarctic stratosphere in October and November, replenishing lost ozone before the most intense sunlight reaches the southern hemisphere. Because this process was delayed, ozone remained diminished even into summer. While polar regions normally receive far less intense light than the midlatitudes, the amount of ultraviolet light reaching Palmer Station last year surpassed the peak summer levels typically seen in a city like Chicago. □

Vasodilating drugs may help – and harm

George often suffers ischemia, reduced blood flow to the heart that is triggered in part by atherosclerosis, fatty deposits that can clog coronary arteries. When George feels the chest pain caused by an ischemic attack, he reaches for nitroglycerine, a drug that dilates the coronary arteries and thus floods the heart with oxygen-rich blood.

Although vessel dilators are a common treatment for the millions of Americans who suffer ischemic chest pains, some researchers now suspect that frequent use of such drugs may pose a hidden danger: While the rush of blood relieves chest pain, it also creates free radicals, chemically reactive molecular fragments that often contain oxygen. These fragments may damage the DNA in mitochondria, tiny factories within cells that generate energy. After repeated attacks, the mitochondria may lose some of their capacity to produce the energy that fuels the heart's pumping.

To test the hypothesis linking mitochondrial-DNA damage to heart disease, Douglas C. Wallace of Emory University in Atlanta and his colleagues obtained cardiac tissue from autopsies or heart-transplant operations. The team examined heart tissue from seven people with severe atherosclerosis and 10 with other types of heart problems. Tissues from 10 people with no evidence of heart disease served as controls.

In the Oct. 2 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, the researchers report finding no detectable damage to mitochondrial DNA in hearts from controls under age 40, and only very low levels of this DNA injury in tissues from controls over 40. Some mitochondrial-DNA damage – perhaps caused by brief periods of reduced blood flow to the heart – may therefore represent a normal part of aging, Wallace says.

More importantly, when compared to controls, all hearts obtained from people with atherosclerosis showed an eight- to 2,200-fold increase in mitochondrial-DNA damage. This suggests hearts experiencing regular ischemic attacks may suffer irreversible mitochondrial damage that in turn leads to a decline in the heart's pumping ability, the researchers say. Because three of the 10 people with other cardiac abnormalities also possessed mitochondrial-DNA damage, Wallace's group suspects mitochondrial-DNA injury may play a role in other types of heart disease as well.