Behavior

Mass hysteria mars the music

On April 13, 1989, nearly 600 students from three junior and senior high schools tuned their instruments and cleared their voices for the 40th annual "Stairway of the Stars" concert in Santa Monica, Calif. No sooner had the assembly commenced than headaches, dizziness, weakness, abdominal pain and nausea spread among the student performers. About half the students — but no one in the audience — developed some combination of these symptoms; 16 girls in the soprano section of the chorus fainted. Officials quickly evacuated the auditorium and ambulances rushed 19 students to hospitals.

A survey of most of the students in the ill-fated orchestra, conducted three weeks after the episode and described in the September AMERICAN JOURNAL OF PSYCHIATRY, indicates they fell prey to "mass hysteria" — not the sort exhibited by fans at a rock concert, but the sudden appearance in a group of temporary physical symptoms stemming from psychological causes. Researchers have previously noted several other outbreaks of mass hysteria among schoolchildren and adults. Numerous physical and emotional factors contribute to these occurrences, but in Santa Monica, social transmission played a key role, maintain psychiatrist Gary W. Small of the University of California, Los Angeles, and his colleagues. Illness most often struck those students who first observed a friend with symptoms, Small's team reports.

Of the 519 student performers who completed a questionnaire developed by the researchers, 247 reported a sudden illness at the concert. Chorus members from one school, particularly girls in the soprano section, experienced the highest rate of symptoms. Social transmission exerted the strongest effects in those youngsters, the researchers contend.

The survey also found that girls suffered a higher rate of illness than boys — 51 percent compared with 41 percent. One-quarter of the symptomatic students had a chronic medical illness, most often asthma, that might have contributed to hysterical reactions, the researchers note.

A clear trigger for the brief incapacitation of the student orchestra evades investigators, "although the psychological stress of performance anxiety probably contributed to symptoms," Small's group concludes.

The educated IQ

In the contentious field of intelligence testing, some researchers argue that IQ scores represent a measure of stable, general intelligence that underlies achievement at school and work. But a review of nearly 200 studies charting IQ development indicates that IQ rises as people spend more time in school, regardless of the quality of schooling, according to a report in the September DEVELOPMENTAL PSYCHOLOGY.

Even the most basic schooling fosters thinking and problem-solving skills tapped by most IQ tests, asserts psychologist Stephen J. Ceci of Cornell University in Ithaca, N.Y.

Ceci notes several trends in the data: Small but consistent IQ drops occur during summer vacation, especially among youngsters living in poor areas; children who attend school intermittently experience steadily declining IQs; children who begin school late or who drop out have lower IQs than their peers; fluctuations in IQ scores closely parallel peaks and valleys in academic achievement scores, suggesting that both measures respond to similar school influences; and average IQs rose dramatically from 1952 to 1982 in 14 industrial nations (SN: 8/15/87, p.108) as the average level of education for citizens in those countries increased.

Other factors, including genetics, affect individual IQs, Ceci acknowledges, but the studies suggest that the magnitude of the educational effect ranges from losing one-quarter of an IQ point to six IQ points per year of missed school.

Biotechnology

Gene fix for muscular dystrophy defect

One out of every 3,500 boys born worldwide lacks a functional gene for the muscle protein dystrophin. The deficiency leads to muscular dystrophy, a progressive muscle wasting that begins in childhood. Boys with a complete lack of dystrophin — who have a severe form of the disease, known as Duchenne's muscular dystrophy — usually die in their early 20s from suffocation or heart failure.

Researchers seeking a genetic cure for muscular dystrophy have now successfully inserted normal copies of the human dystrophin gene into the muscles of mice born without the critical protein. Although they concede that "the efficiency of this gene-transfer technique needs to be increased before it can be used clinically," they say their achievement holds out hope for an eventual genetic fix for muscular dystrophy.

Using technology developed at Vical Inc. in San Diego, the researchers injected the thighs of the deficient mice with tiny circlets of DNA, called plasmids, containing the dystrophin gene. They report in the Aug. 29 NATURE that roughly 1 percent of the thigh-muscle cells in the mice took up the new genes and made dystrophin, which the researchers detected using labeled antibodies. Moreover, the dystrophin was located in its proper position on the cells' membranes.

"The dystrophin protein was in fact produced by the introduced genes, and was found in places where it's usually seen in normal muscle," says study director Jon A. Wolff of the University of Wisconsin-Madison.

However, when the team injected the same plasmids into the heart muscles of mice, dystrophin appeared in only a handful of isolated clumps. Wolff and his co-workers suggest that inserting new genes into the heart is more difficult because heart muscle cells, unlike those of skeletal muscle, do not fuse together to form large fibers that share proteins.

Last year, a team led by Peter K. Law of the University of Tennessee in Memphis pioneered a cell-transplant approach to treating muscular dystrophy (SN: 6/16/90, p.380). Law's group injected a patient's big toe with immature muscle cells taken from the arm of his father. These cells, called myoblasts, fused with the boy's big-toe muscle and produced dystrophin.

Terence A. Partridge, who first demonstrated the cell-transplant technique in a 1989 experiment with mice, calls Wolff's gene-transfer approach "superior." But myoblast transplants are "far more effective" for the time being, he asserts in an editorial accompanying the new report, because an injection of 100,000 cells can yield dystrophin production in 30 to 40 percent of muscle fibers, spread over a large area. Partridge is a histopathologist at Charing Cross and Westminster Medical School in London, England.

FDA panel backs septic shock treatment

A committee of outside experts convened by the Food and Drug Administration has endorsed a human antibody, HA-1A, for the treatment of life-threatening bacterial infections.

The panel decided this month that the drug is safe and effective in quenching the microbial poisons that can kill people infected with gram-negative bacteria. Such bacteria thrive in patients with suppressed immune systems, such as those undergoing chemotherapy. The microbes make endotoxins that can cause the organ failure of septic shock.

HA-1A binds to endotoxins, blocking their effects. Earlier this year, researchers reported that the antibody reduced deaths by 39 percent among a group of 200 patients with serious gram-negative infections (SN: 2/16/91, p.100).

Centocor, Inc., of Malvern, Pa., makes HA-1A by culturing genetically engineered human cells in the laboratory. Xoma Corp. of Berkeley, Calif., is using mouse cells to develop a similar drug.