Presumed mute, liquid crystals sound off

The springs, the jewels, and the tiny wheels are gone, but a ghost of the ticks and tocks of traditional clocks may linger inside today's digital watches, a new and surprising discovery suggests.

In fact, an imperceptible murmur may emanate from cell phones, calculators, and other devices with liquid-crystal displays.

"It's an amusing thing," comments Robert B. Meyer of Brandeis University in Waltham, Mass. "People will think, 'My liquid crystal is actually chirping at me.'"

A liquid crystal is a fluid of rodlike molecules that line up in an orderly way. Manipulating the alignment can affect the fluid's transparency (SN: 8/7/99, p. 87; 6/1/96, p. 348). In most liquid-crystal displays, layers of glass coated with thin, transparent electrodes sandwich a film of oil-like liquid crystal about 10 micrometers thick. Rods of the liquid crystal normally lie stacked in planes parallel to the glass surface, a horizontal orientation that allows light through the cell. Application of a voltage flips the molecules to a vertical position, turning the cell opaque enough to appear dark.

Ordinarily, no one hears the newly reported chirps because everyday liquid-crystal displays use low voltages and their components are probably not the best size to make loud sounds, says Jay S. Patel of Pennsylvania State University in State College.

Young Jin Kim, a researcher in Patel's lab, chanced upon the sounds while testing an experimental optical switch, or cell, that operates at more than 30 volts, much higher than the few volts of most displays. With each voltage pulse, the switch gave off a click "like the sound of a ratchet," Kim says. A voltage varying as a sine wave transformed the click into a steady hum.

In search of the sounds' cause, the incredulous scientists made and tested hundreds of cells, using a sensitive microphone and amplifiers. They concentrated on nematic liquid crystals, the most widely used type of liquid-crystal material.

To confirm that the liquid-crystal rods themselves make the sound, the researchers applied voltages to cells containing disordered liquids. Then, they didn't hear a peep. They also heated the liquid crystal above a temperature at which orderly liquid-crystal behavior breaks down. Again, no sounds.

They performed other tests as well. "From so many studies, we can confirm that the sound is from the motion of liquid-crystal molecules," Kim says.

The experiments demonstrate electrical control over both optical and acoustic properties of a cell, remarks Peter Collings of Swarthmore (Pa.) College. "That's why I see some cause for excitement," he says.

Patel says that it's too early to tell, however, whether the unexpected effect might lead to novel practical applications for liquid crystals.

He and Kim speculate that the sloshing of molecules as they flip from horizontal to vertical and back somehow vibrates the glass. The container, in turn, emits sounds at frequencies from 1 to 14 kilohertz—within human hearing range. Sound volume peaks when molecules flip at natural vibration frequencies of the containers, the researchers found.

How such weakly powered motion could agitate the glass is "a mystery," Meyer says. "It begs some kind of analysis."

The researchers report their discovery in the Sept. 27 APPLIED PHYSICS LETTERS. They claim there that until now, "there have been no reports of sound generation from liquid crystals" due to a changing electric field.

However, Antal Jakli of Kent (Ohio) State University disputes their claim. He cites more than 20 publications since 1985, many of which he coauthored, that describe similar effects in the types of liquid crystals known as smectic and ferroelectric.

Patel admits that his group was unaware of the work of Jakli and his colleagues. Nonetheless, for the widely used nematic liquid crystals that his group has investigated, "nothing like this has ever been done before," Patel asserts. —*P. Weiss*

Physicians find clues to vision deterioration

Blindness in the elderly often stems from a disease called age-related macular degeneration. Yet the cause of this affliction remains unknown.

The disease damages an area, called the macula lutea, at the center of the retina. The retina comprises the tissue at the back of the eye that converts optical images to electrical impulses, which then travel to the brain via the optic nerve.

A new theory suggests that some age-related macular degeneration results from overeager repair of mild injuries to retinal cells. Moreover, high fat concentrations in the bloodstream may exacerbate this immune overreaction. Another line of research suggests that the disease may result from constricted blood flow and gradual starving of retinal cells.

Scientists know that age-related macular degeneration can attack the retina in two ways. The so-called dry form results when plaquelike deposits appear under the retina's outer layer. The plaque damages vital light-sensing cells, and as these die off, vision—especially in the center of a person's field—fades.

The so-called wet form of the disease is less common but more likely to cause blindness. It arises when extra blood vessels form in an outer layer of the eye called the choroid. These vessels invade the retina and leak fluid into it. The assault kills light-sensing cells and forms scar tissue.

Scientists at the University of Miami School of Medicine have new evidence that cellular repair runs amok in the dry form of the disease. After mild damage, such as that caused by a bright light, retinal cells in a test tube didn't appear to heal properly. They shed some of their cellular material into blebs, or small sacs of fluid, between the outer layers of the retina, reports ophthalmologist Scott W. Cousins. He and his colleagues spotted the new blebs after the team engineered retinal cells to glow fluorescently in a laboratory dish.

The blebs appear to trigger an immune overreaction. In a separate experiment, scavenging cells called macrophages from about one-third of study participants with the dry form of age-related macular degeneration showed an overresponse when they came into contact with blebs, spurring an inflammatory reaction, Cousins says.

In some patients, macrophages may rush to the scene of a mild injury and release toxins "that make the damage worse," Cousins suspects.

In studies on mice, Cousins' team also found that high-fat diets made old mice prone to bleb formation, whereas feeding them vitamin E helped prevent it. Destructive chemical groups called free radicals that are fueled by fats may induce blebbing, Cousins says. "Essentially, we're suggesting that these kinds of toxins can be looked at as potential trigger mechanisms for the deposit formation of macular degeneration," Cousins concludes. He presented the findings this week at a meeting in Universal City, Calif., sponsored by Research to Prevent Blindness.

Also at the meeting, researchers from the University of Pennsylvania in Philadelphia reported that some patients with the dry form of the disease have lower-than-normal blood flow to the retina. Poor circulation fails to deliver nutrients and does a poor job of clearing away existing cellular waste products from the area between the retina and choroid, says ophthalmologist Juan E. Grunwald.

By shining laser light into the eyes of healthy people, Grunwald's team noted that the older participants had only about two-thirds the blood flow from the choroid as the younger volunteers did. Examination of patients with dry age-related macular degeneration showed an even greater decrease in blood flow with age.

Patients with the dry form of the disease are more likely to develop the wet form than healthy people are, and this difference may be due to the poor circulation, Grunwald says. His team is now investigating that possibility. —*N. Seppa*