

The way to really cold logic

If certain recent developments at Bell Laboratories are an augury of the future, Silicon Valley may be turned into Josephson Junction. An article in the May-June BELL SYSTEMS TECHNICAL JOURNAL by T.A. Fulton and L.N. Dunkleberger of Bell Labs reports successful fabrication and operation of a data processing chip that uses Josephson junctions as the operative elements in its circuitry.

A Josephson junction consists of two metal layers separated by a very thin insulating strip. When this arrangement is chilled to the point that the metals become superconducting, the relation between current and voltage across such a junction has a number of singular characteristics. These properties permit use of Josephson junctions in the same ways as transistors are used in computer circuitry.

Operating at the temperature of liquid helium (4 kelvins), Josephson junction circuitry uses less power, works faster and generates far less heat than silicon circuitry. According to a Bell Labs announcement, a Josephson junction computer the size of a baseball could process the same amount of information as a room-sized silicon computer and do it faster.

Technical difficulties that have stood in the way of applying this technology in-



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clude fabrication of the junctions, refrigeration and connection of the superconducting circuitry to the outside world, which is at room temperature. The chip displayed in the Bell Labs announcement is a parallel multiplier intended for use in processing of color television images. It consists of 548 Josephson junctions connected in pairs with a resistor across each pair. The pairs are called JAWS, Josephson Atto-Weber Switches, because of resemblance to a shark's face. (An atto Weber is a unit used for measuring extremely small amounts of magnetic flux.) The chip functions in a vessel of liquid helium the size of an umbrella stand, and it can talk to silicon circuitry at room temperature. This chip, although speedy enough for the intended image processing, is not as fast as Josephson junction technology can go. The next step is to make smaller, faster chips for other applications. □

Loud noise may confuse the brain

Loud noise can damage the ear — that has been well established. But now James Willott and Shao-Ming Lu report in the June 18 SCIENCE on animal research demonstrating that "moderate" noise can affect individual brain cells as well. The Northern Illinois University scientists believe their findings with mice may offer an explanation for understanding such human afflictions as "ringing in the ears" and hearing problems where sound comprehension—not audibility—is impaired.

Microelectrodes were implanted in the brains of anesthetized mice, and positioned so that the voltage produced by the firing of an individual neuron (nerve cell) could be isolated — and recorded — against background levels of electrical activity. Actual sound exposures lasted a mere 30 seconds. Yet the moderately loud, 95-110 decibel broadband noise was sufficient to induce changes in the behavior—firing pattern—of monitored neurons.

"Responses of the individual brain cells

were often very unpredictable, sometimes paradoxical," Willott told SCIENCE NEWS. For instance, a temporarily or partially deafened auditory system should be less responsive than normal to sound. "Yet despite the fact that some hearing loss was induced," he noted that "the excitability of brain cells actually increased."

Sounds travel through the ear by vibration. Upon reaching the inner ear, these vibrations actually bend sensitive hair cells, stimulating nerves at the hair cells' roots to transmit electrical signals to the brain. It is the brain's function to decipher these signals into what we "hear" or *think* we hear. But if somewhere along the signal path the transmission is altered or garbled, the brain may have difficulty or actually err in deciphering what sounds had actually entered the ear.

Willott and Lu studied brain neurons responsive to auditory signals. How a neuron responds to sound can vary. For instance, one might fire throughout the

duration of a given sound signal; "It seems to be coding the presence of sound simply by firing repetitively for as long as the sound is on," Willott explains. In contrast, others may exhibit an onset response. They fire once, when a sound commences, then sit quietly, waiting to announce the beginning of a new sound.

"Presumably, a neuron that gives an onset response to sound is doing something quite different from one which gives a sustained, repetitive response," Willott says. Yet after exposure to loud noise, some of the neurons that the Northern Illinois team were monitoring changed from firing in a sustained manner to adopting the onset response, and vice versa. "What precisely the implications of that are on coding we don't know, because we don't understand coding," Willott says. "But it does indicate that there's an alteration in the fundamental activity of given neurons. One might guess that it confuses the brain in some way." Willott adds that in a few cases where he was able to watch individual neurons for prolonged stretches, "there was some recovery"—meaning the neurons eventually reverted back to what he termed their normal firing pattern.

The scientists observed another important change: After inducing partial deafness in their mice, some of the monitored neurons actually responded more vigorously. (For humans, the stimulus used "would probably be fairly comparable" to sounds emitted by a passing freight train or jet plane, Willott says.)

"Many neurons, when nothing is happening, slowly fire at a spontaneous rate," Willott explains. Listening to certain frequencies can increase the firing rate, while other frequencies can slow it, sometimes to a dead stop. The firing rate seems to be controlled by two influential forces—one acting to excite or speed it, the other acting to inhibit or slow it.

The scientists speculate that by inducing a hearing loss, they have altered the central nervous system's accuracy in coding electrical signals meant to describe the acoustic environment.

"Let's say that high-frequency sound normally [inhibits the firing of] an individual neuron," Willott says. "We blast the animal with sound and knock out the high-frequency sensitivity of its inner ear. Now that same neuron can no longer be stimulated by high frequencies and the inhibitory input to the neuron can no longer be activated." As a result, the hearing loss produces an effect — excitation of the neuron's firing rate — totally opposite to what might have been expected from the sound input.

"Who knows," Willott says, "the effect of this might be a false impression that there's sound" when there is none. Tinnitus—a perceived ringing in the ears—has been linked with damage to the inner ear. "But data in the SCIENCE paper suggests that maybe the brain could be responsible," Willott says. —J. Raloff