Simple structures not crystal clear

For years, textbooks of solid state physics have taught students to calculate the structure of crystals. Physicists tended to believe that they were quite familiar with the structure of the simplest crystals. Now comes Barry L. Berman of the Lawrence Livermore National Laboratory in Livermore, Calif., to say that studies with a new method of probing crystal structure — channeling radiation — show serious disagreements with the standard calculations and indicate that physicists do not know as much as they think they do about even the simplest crystals.

Channeling radiation is a somewhat difficult technique to apply. It is only a few years since Berman and his colleagues demonstrated its existence (SN: 5/12/79, p. 311). It is electromagnetic radiation emitted by electrons and positrons as they move at relativistic speeds through crystals in paths guided by the structures of the crystals. Electrons or positrons struck against a solid will usually be absorbed. If they are introduced in just the right way, however, they will escape absorption and "channel" their way through the crystal. Positrons channel between the planes of atoms in the crystal. Electrons channel along the planes, or, rather, weave around them in a pattern that prompts Berman and colleagues to refer to electrons as "weavons." The moving electrons and positrons feel electric forces that undulate, being stronger near the ions of the crystal lattice and weaker away from them. So the moving particles wiggle from side to side, and as they do they emit electromagnetic radiation.

Berman's group first demonstrated channeling radiation in silicon. They went on to diamond, which has the same crystal structure as silicon, and then to a compound, lithium hydride. The first diamond experiment, with artificial diamond, took place at the Saclay Laboratory in France, where Berman spent a year. Back in Livermore, Berman took up natural diamond and lithium hydride. Three papers presented at the recent meeting in San Francisco of the American Physical Society describe this work. Co-authors include Sheldon Datz and R.W. Fearick of Oak Ridge National Laboratory; J.O. Kephart, R.K. Klein, R.H. Pantell, H. Park and R.L. Swent of Stanford University; M.J. Alguard of Measurex Corporation and M.V. Hynes of Los Alamos National Laboratory.

Natural diamond was studied in two forms, relatively pure and containing dislocations known as nitrogen platelets. If nitrogen atoms are present in a diamond, they will arrange themselves in little plates that insert themselves between planes of carbon atoms and push the carbon planes out of their usual location. A three-dimensional crystal can be approached from three orientations, along the planes that crystallographers designate $\{100\}$, $\{110\}$ and $\{111\}$. Electrons and positrons will channel along any of these orientations, and as the two kinds of particles channel differently, that gives six different probes of crystal structure.

The experimenters were able to predict how the platelet dislocation would affect channeling along the different orientations, whether the passage of electrons or positrons would be diminished or completely suppressed. Measurement supported their predictions, and so they believe they have a good method for describing dislocations in crystals, one that could be useful in materials science and such things as quality control in Silicon Valley.

However, significant discrepancies with the standard way of calculating crystal structure appeared in natural diamond. Channeling in the $\{100\}$ and $\{110\}$ orientations agreed with standard calculations; those in the $\{111\}$ didn't. In one and the same crystal, two planes agree, one doesn't. "We don't understand it," says Berman.

In lithium hydride things were worse: for $\{100\}$ really bad discrepancy, for $\{110\}$ moderate discrepancy, for $\{111\}$ "right on." Berman stresses that this is a simple ionic crystal with a cubic structure like that of rock salt. It should be one of the easiest to calculate. He concludes: "We do not understand the crystalline potential for this very elementary crystal."

—D.E. Thomsen

Scanning senility: A local problem?

In 1906, the German pathologist Alois Alzheimer diagnosed the first case of what is today called Alzheimer's disease, a form of dementia associated with two specific signs of brain damage: so-called "tangles" and "plaques" in the brain's nerve fibers. Today, Alzheimer's disease can still be diagnosed conclusively only if an autopsy reveals this conspicuous brain damage, but researchers have yet to explain how these brain abnormalities lead to intellectual deterioration.

The best theory to date has involved the plaques, which scientists believe are corpses of neurons that, before dying, carried a specific chemical transmitter, acetylcholine, from deep in the brain to stimulate the thought and memory centers in the frontal region of the brain. A government scientist is now challenging the importance of the acetylcholine connection, arguing from new data that there must be another unidentified neurotransmitter that is primarily responsible for the dementia of Alzheimer's disease.

Thomas N. Chase, a researcher at the National Institute of Neurological and Communicative Disorders and Stroke in Bethesda, Md., used a positron emission tomography (PET) scanner to study brain metabolism in 20 subjects clinically diagnosed as Alzheimer's victims. As he reported at a seminar last week, the average Alzheimer's patient was metabolizing 28 percent less glucose than normal, suggesting significantly diminished activity in the brain's cortex. Interestingly, however, the decreased fuel metabolism was not seen uniformly throughout the cortex, Chase reported. The rear of the brain — especially the posterior parietal lobe—showed a deficit of as much as 40 percent, while the frontal area was relatively spared.

What this means, according to Chase, is that the parietal lobe — also known as the association cortex, because it is responsible for integration of sensory inputs — bears the brunt of the damage in Alzheimer's disease. The same imbalance was found in both the severely and the mildly demented subjects, and in fact the level of impairment in individual subjects (as measured on a battery of memory and reasoning tests) closely matched the decrease in parietal activity — not the decrease in overall brain metabolism.

These results, Chase suggests, run contrary to the established view that Alzheimer's dementia results from generalized brain damage. If the acetylcholine deficit were the cause of dementia, the plaques, like the glucose deficit, would primarily be seen in the rear cortex. But the plaques are diffuse, perhaps even predominant in the frontal areas of the brain — suggesting to Chase that this abnormality, while indisputably present in the disease, cannot explain the symptoms of dementia. Furthermore, he notes, clinical studies using drugs that increase acetylcholine levels have produced marginal therapeutic results at best.

Chase believes that there must be another neurotransmitter malfunction involved, and this malfunction must be localized in the association cortex. It need not be a chemical that is present only in that brain region, and indeed no such transmitter is known to exist, he says; it might instead be that a common neurotransmitter functions abnormally in the parietal area.

According to Joseph T. Coyle, a Johns Hopkins University neuroscientist who has done some of the major work on acetylcholine, studies now underway are looking for additional brain chemicals that might be involved in Alzheimer's disease. There is already evidence that two, somatostatin and norepinephrine, are abnormal in some patients, he notes. No scientist studying Alzheimer's disorder would argue that the acetylcholine system is the only system involved in the disorder, Coyle says; but the defect is pointing to critical brain stem systems that may regulate cerebral function and most likely contribute to the dementia. *—W. Herbert*