response to their internal associations — bonds, repellencies and long-distance, unbonded attractions. They then fed these "force field parameters" into a computer and instructed it to apply this code of conduct to the novel structures they began designing.

Once the team defined a new porphyrin's composition, the computer predicted the hypothetical molecule's preferred configuration. Normally, a porphyrin is flat. But these predictions indicated that as the scientists began replacing hydrogen atoms at the periphery of the molecule with relatively large and bulky subgroups — such as phenyl rings — the entire structure would ruffle. Some versions contorted so severely that a deep pocket formed around the metal atom at the center of the catalyst.

One catalyst formed a pocket "just the right size to hold carbon dioxide — and not something else," notes Shelnutt. By altering electric charges on the pocket's lining, "We can switch the catalyst's potential affinity from carbon dioxide to other small molecules like methane."

How good were those predictions? Shelnutt collaborated with Kevin M. Smith at the University of California, Davis, to synthesize samples of his newly designed porphyrins. X-ray crystallography and Raman spectroscopy have just confirmed that the actual compounds match his predictions. Indeed, Shelnutt's predicted structures are "embarrassingly accurate," says Sandia chemist Alan P. Sylwester — so close, he jokes, that they look as if they were traced from structural diagrams of the synthesized compound.

Shelnutt's team will continue working toward catalysts that might make something useful out of some of the uncontrolled carbon dioxide that poses a serious threat of global warming. The researchers are also designing a solar-driven process to detoxify chemically contaminated water. In preliminary tests, another novel porphyrin catalyst appears more than 100 times faster than the titanium-dioxide catalyst currently used in a similar solar decontamination system now under development. — J. Raloff

Neandertals to investigators: Can we talk?

European Neandertals, who lived from about 130,000 to 35,000 years ago, possessed all the anatomical tools needed for speaking as modern humans do, according to a report presented at the annual meeting of the American Association of Physical Anthropologists in Las Vegas last week.

The new analysis of Neandertal and modern human skulls, conducted by David W. Frayer of the University of Kansas in Lawrence, enters a debate over Neandertal vocal capacities that began in the 1970s. Arguments intensified recently with the discovery of a small neck bone said by its discoverers to demonstrate a fully modern facility for speech among Neandertals (SN: 7/8/89, p.24).

"Neandertal speech and language ability was equivalent to ours," Frayer maintains. "Whether they indeed did speak is another issue."

Frayer studied the degree of bend in the base, or basicranium, of Neandertal and modern human skulls. A flat basicranium — ubiquitous in nonhuman animals — indicates that the larynx, or voice box, sits high in the neck. An arched cranial base signifies a lower larynx and a vocal tract capable of producing the sounds of modern human speech.

Often, important features of the basicranium are poorly preserved on ancient fossils. In his study, Frayer relied on a measurement of the angle from a relatively easily determined point near the center of the basicranium to a point at the front of the upper jaw.

The extent of basicranial flattening in four European Neandertal specimens falls within the range observed in a

sample of modern human skulls dating from 25,000 years ago to medieval times, Frayer contends. In fact, some of the older modern skulls display flatter skull bases than the Neandertals, he says. The evidence supports theories of a close evolutionary link between Neandertals and modern humans, he adds.

One of the Neandertal skulls studied by Frayer was reconstructed in 1989 by a French anthropologist who also argued that the angle of its basicranium falls within the range of modern humans.

Other researchers, led by anatomist Jeffrey T. Laitman of Mount Sinai School of Medicine in New York City and linguist Philip Lieberman of Brown University in Providence, R.I., discern a flatter cranial base and more restricted speech ability in European Neandertals than in modern humans. Laitman's group estimates the position of several anatomical markers on fossils to determine four basicranial angles from the back of the head to the jaw; Lieberman devised a computer model of the Neandertal vocal tract based on the skull that was later reconfigured by the French investigator.

Although Neandertals had the ability to vocalize, their speech quality fell short of that exhibited by modern humans, Laitman asserted at the Las Vegas meeting. "I'd advise caution in measuring only one angle on the basicranium, as Frayer did," he says.

Frayer cites the poor preservation of basicranial features as the prime reason for using his study method. "I'm uncomfortable with how much of the cranial base is missing on Neandertal specimens," he remarks.

— B. Bower

Gene determines when cells live or die

When an amphibian makes the leap from tadpole to toad, the cells in its tail kill themselves because they're no longer needed. Biologists call this phenomenon programmed cell death, or apoptosis. It's a normal part of animal development, but no one knows how it works.

Now, researchers studying roundworms have uncovered a clue to the mystery. A cell's fate, they find, teeters on a single gene that keeps the built-in suicide program from starting up.

"The observation that lots of cells in the animal need something in them to continuously protect them from dying is very intriguing. It's the first time it's ever been shown that such a thing could exist," says molecular biologist Ronald E. Ellis of the University of Wisconsin-Madison.

Working at the Howard Hughes Medical Institute at the Massachusetts Institute of Technology in Cambridge, Ellis and his colleagues studied the roundworm Caenorhabditis elegans and found that a gene called ced-9 acts as a switch to regulate programmed cell death. In C. elegans mutants with the ced-9 gene turned on, cells that normally would have died during the animal's development survived instead. Conversely, in roundworms with a mutation that turned off the gene, cells that normally would have lived committed suicide, the researchers report in the April 9 NATURE.

With ced-9 turned off, "cells that were supposed to survive and become neurons or muscles or some other kind of cell were killing themselves," says Ellis. "The animals eventually died."

Although the exact mechanism underlying this process remains unclear, ced-9 appears to control two other genes, ced-3 and ced-4, previously found to direct a roundworm cell's suicide process. Ellis and his co-workers discovered that ced-9 had no effect in mutant roundworms lacking these two genes. "The switch doesn't matter if there's no machine at the other end," Ellis explains.

While *C. elegans* consists of a mere 1,090 cells, biologists believe that studies of programmed cell death in this simple model will help them understand how the process works in more complicated animals, including humans. In fact, ced-9 seems to parallel a gene called bcl-2, which controls the suicidal tendencies of B- and T-cells in the human immune system, says Stanley J. Korsmeyer of the Washington University School of Medicine in St. Louis, who studies apoptosis in humans.

"Bcl-2 has implications for how ced-9 may act," Korsmeyer says, "while ced-9 has implications for the regulation of cell death in mammals." -M. Stroh

SCIENCE NEWS, VOL. 141