the May 16 NATURE.

The newly discovered animal "makes the Cambrian bestiary look a lot less bizarre than it used to," says Stefan Bengtson, a specialist in Cambrian fossils at Uppsala University in Sweden.

The keystone fossil has a segmented body stretching 5 to 6 centimeters in length, with 11 pairs of balloonish legs that end in two-pronged claws. Onychophorans share these same features, leading Ramsköld and Hou to group the new fossil with the existing phylum. In the modern world, onychophorans live on land, mainly in the moist litter of tropical forests. Bengtson describes them as looking like a cross between a centipede and the Michelin Man.

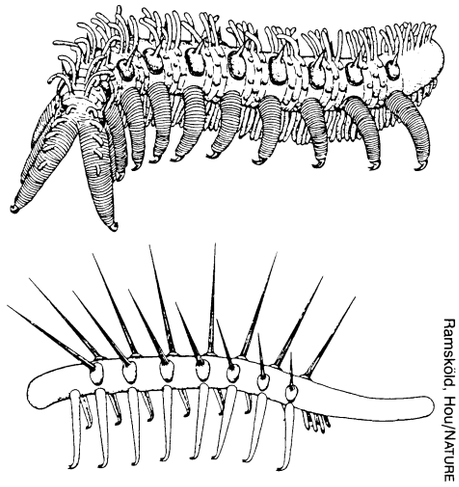
To make the onychophoran connection, Ramsköld and Hou have stretched the boundaries of the phylum. The Cambrian animal, unlike the modern forms, lived in the ocean and sported 10 sets of rigid back plates, each topped by a pointy spine.

The two researchers have used the new fossil as a stepping stone to categorize some of the most puzzling Cambrian fossils found in Canada's Burgess Shale formation and other sites around the world. One of the weirdest, appropriately named *Hallucigenia*, looks like something out of a Dr. Seuss book. Previous restorations have depicted this inch-long animal walking on seven pairs of spiky legs, with a row of seven tentacles sprouting from its back – a beast so bizarre that it defies categorization.

But Ramsköld and Hou think the standard reconstruction of *Hallucigenia* has got the animal turned upside down. They suggest that the tentacles are actually legs, and the spikes stick up from the back of the creature, like longer versions of the spines seen on the new Chinese fossil. The connection between *Hallucigenia* and the unnamed Chengjiang animal has led the two researchers to draw *Hallucigenia*, as well as some other Cambrian oddities, into the fold of Onychophora.

Although this work has attracted the attention of other Cambrian experts, most paleontologists remain unconvinced by Ramsköld and Hou's interpretation of *Hallucigenia*. Their biggest hurdle is a missing set of legs. Only two good *Hallucigenia* fossils have been found so far, and each shows only one row of the "tentacles" that Ramsköld and Hou have labeled legs. The two researchers believe another set lies buried beneath the visible legs.

"I think they're probably right. It may well be that the animal is the other way up," says paleontologist Simon Conway Morris of the University of Cambridge in England. If so, the reversal will overturn Conway Morris' own interpretation of *Hallucigenia*, which he proposed in the late 1970s.



Ramsköld, Hou/NATURE

Newly discovered Cambrian fossil (top) and revised view of Hallucigenia.

This week, Conway Morris and Ramsköld visited the Smithsonian Institution in Washington, D.C., and examined the two *Hallucigenia* specimens housed there, in hopes of resolving the issue.

– R. Monastersky

AIDS dementia: Neurons nixed by virus?

Almost one-third of all AIDS patients eventually develop encephalitis, or inflammation of the brain. As their encephalitis progresses, memory loss and other forms of dementia become increasingly severe. This has led scientists to suspect that AIDS dementia arises because the inflammation damages brain cells.

Now, evidence from an autopsy study suggests a more direct mechanism for these debilitating changes: The AIDS virus itself may destroy neurons in the frontal cortex of the brain.

Laboratory studies in recent years have shown that the AIDS virus, HIV, somehow manages to kill neurons in culture, even though it does not enter and infect them. Those findings spurred three British neuropathologists to determine whether the virus can, in the absence of encephalitis, inflict enough neuronal damage to cause dementia in people with AIDS. Ian P. Everall and his colleagues at the Denmark Hill Institute of Psychiatry in London tested their hypothesis by examining the frontal cortex, an area involved in thought and reasoning, of 11 deceased AIDS patients and eight people who died of causes unrelated to AIDS or encephalitis.

The investigators used a newly developed tool called a dissector to calculate the density of neurons in three-dimensional sections of the frontal cortex samples. The neuron density in the AIDS patients' brains was almost 40 percent lower than in the non-AIDS brains, they found.

Although fluid from all of the AIDS brains harbored HIV, six of the 11 showed no signs of encephalitis. Moreover, the researchers discovered that the neuron density in those six brains was just as low as in the five encephalitic AIDS brains. This, they say, suggests that HIV alone can cause a loss of neurons.

Because Everall and his co-workers did not have access to the patients' clinical records, they do not know whether any of them actually had dementia. Nonetheless, they assert in the May 11 LANCET, "the cause of dementia in AIDS patients has to be reevaluated."

"This is the first investigation that has shown neuronal loss without HIV encephalitis," says coauthor Peter L. Lantos. Finding such a dramatic loss in a cortical area of the brain is especially significant, Everall adds, because a comparable degree of cortical cell loss is associated with the severe dementia suffered by Alzheimer's patients.

"These types of losses are going to have a physiological impact," says Clayton A. Wiley, a pathologist at the University of California, San Diego.

Because the new study does not establish any clinical connection with dementia in encephalitis-free AIDS patients, Everall cautions that the findings do not prove that HIV causes dementia by killing neurons. He does, however, propose what he calls "a sensible and logical mechanism" by which the virus might kill cortical neurons directly. An HIV envelope protein called gp120 may interfere with a brain peptide called VIP that some cortical neurons need in order to send electrical signals to one another, he told SCIENCE NEWS. Researchers already know that this peptide can, in the test tube, protect brain neurons from the toxic effects of gp120, he adds.

Brynmor A. Watkins of the National Cancer Institute in Bethesda, Md., questions the British team's exclusive focus on the frontal cortex, which may have led them to overlook encephalitis in other brain parts, especially lower regions such as the basal ganglia and the cerebellum. Encephalitis in these subcortical regions correlates strongly with certain symptoms of AIDS dementia, such as limb weakness and poor coordination, he says. In addition, he argues that the six seemingly encephalitis-free AIDS patients may have had frontal-cortex inflammation at one time and that their encephalitis subsided before they died.

– T. Walker