The Moho is immutable no more

For decades, geoscientists have thought of the Moho as a simple, immutable dividing line between the earth's crust and mantle. Discovered in 1909 by Yugoslavian seismologist Andrija Mohorovičić, the continental Moho resides at depths of 35 kilometers, where the speeds of seismic waves traveling through the earth abruptly change.

In recent years, high-quality seismic reflection profiling — in which sound waves sent into the crust bounce back off contrasting rock types (SN: 12/8/84, p.364) — has provided seismologists with a much closer look at the Moho. And they are beginning to see that it has a more complex structure than once thought. This is leading them to wonder exactly what the Moho is, how it is formed and whether it changes with time.

Some of the latest seismic reflection profiles illustrating the puzzling character of the Moho were displayed last week at the meeting of the Geological Society of America in San Antonio, Tex. The data were compiled by the Consortium for Continental Reflection Profiling (COCORP), one of several research groups in the world doing systematic seismic surveys of the crust. COCORP members discussed some of the more than 2,500 kilometers of new seismic lines taken across the northwest Cordillera, the southern Appalachians and the U.S. Basin and Range Province.

One surprising aspect is evident in the Cordilleran mountain belt, which extends from Alaska to Guatemala. Its northwestern section is a patchwork of different crustal fragments that were carried thousands of kilometers by plate motion and jammed into the North American continent. Yet in profiles of the northwestern Cordillera, the Moho shows up as a flat, bright reflection that extends continuously through this region, oblivious to the sutures and different geologic boundaries that are so evident at the surface. Moreover, in some areas of this region, reflection profiles show that fault-like structures dip into the crust but are cut off at the Moho.

"This all implies that the Moho is a young structure," having re-formed after the terrains were plastered on to the continent, says K. Douglas Nelson of COCORP, which is headquartered at Cornell University. Something has made the Moho straight and continuous, erasing the bottoms of older faults and other geologic boundaries; the new Moho was superimposed on the older crustal fabric.

Some sort of change in the Moho's structure is apparent also in comparisons between the Cordillera and younger mountain belts, such as the Himalayas. There, the Moho is discontinuous, jumping to different depths in different regions. How, asks Nelson, does the Moho structure beneath the Himalayas change to that under the Cordillera? What changed the shape and character of the crust?

One possibility is that the newer, continuous Mohos were created — replacing old ones — when the crust was stretched out. In addition to the northwestern Cordillera, sharp, flat and highly reflective Mohos are seen in the Basin and Range Province, the continental shelf around Great Britain, the Paris basin and the U.S. Atlantic shelf. In most of these places, the last known major tectonic event was crustal extension. Perhaps in this process the crustal fabric was smoothed out, thinned and aligned horizontally. Another suggestion, according to Nelson, is that intrusions of magma into the lower crust left horizontal layers that differ in chemical makeup from surrounding rocks.

While the Moho is clearly defined in seismic profiles of these extensional regions, it is much more diffuse in other areas, such as the North American craton — one of the oldest, nonmountainous, "undeformable" parts of the continent. For example, such diffuse Mohos show up in COCORP profiles of flat regions such as Kansas, Minnesota, Wyoming and the Colorado plateau.

"Many geologists have the perception that the processes that made mountain belts in recent times are basically the same processes that made the crust back through the Precambrian," says Nelson. "If that's true, then Precambrian crust in

Balloon use helps heart valves

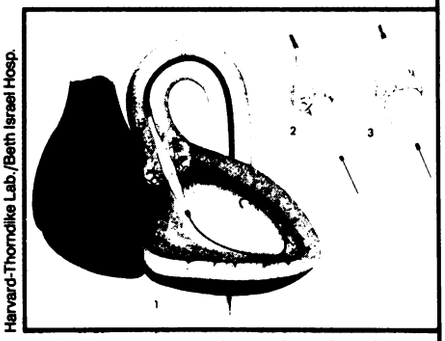
Balloons, first used in hearts to open up narrowed blood vessels, can also free up stiffened heart valves, according to several studies presented this week in Dallas at the American Heart Association meeting. The procedure, known as balloon valvuloplasty, was first done in 1979 in children with congenital valve disease; its more recent application to adults represents a significant broadening of its horizons.

In addition to congenital causes, rheumatic fever and the general aging process can cause valve disease. The four valves in the heart, each several leaves of delicate tissue that jointly open in only one direction, keep blood headed the right way. Calcification of the valves partially fuses the flaps together. Malfunctioning valves can ultimately be deadly.

At the meeting, researchers from Asia, South America, Europe and the United States described positive results in trials of balloon valvuloplasty in dozens of adults. While techniques vary, the basic procedure, first done in adults in 1984, is to thread a balloon through a catheter into a sticky valve while the heart is beating. The balloon is then inflated for up to 40 seconds using a saline/dye solution, at a pressure of 45 to 60 pounds per square inch. The inflation splits the flaps of the valve apart, allowing them to open and close more freely.

William Grossman and his co-workers at Harvard Medical School have done balloon valvuloplasty on 76 people since October 1985. "Results in general have been quite good," he says. Grossman says the two who died within a week succumbed to preexisting damage, not to the valvuloplasty itself.

The current treatment for people with diseased heart valves is either to replace or to surgically slice apart the valve flaps. Tens of thousands of such operations are done annually. But many people with diseased valves are too old or infirm to withstand the surgery. The balloon procedure can be done with painkillers and local, rather than general, anesthesia, and the patient can leave the hospital within a few days, Grossman says.



Harvard-Thorndike Lab./Beth Israel Hosp.

A balloon opens the valve that keeps blood intended for the body from flowing back into the heart. The balloon is inflated with a saline/dye solution at a pressure of 45 to 60 pounds per square inch.

He does not expect the technique to replace valve surgery, however. "This is definitely new and must be regarded as experimental," he says. People with leaky rather than stiff valves will not be helped by balloon valvuloplasty, and the procedure does not keep the opened valve from stiffening up again.

And what if the balloon bursts? It has happened, says Charles McKay of Los Angeles County/University of Southern California Medical Center, where 22 operations — some using two balloons in the same valve — have been done "with very encouraging results." But bursting hasn't been a problem — the deflated balloon is pulled out through the catheter, and the saline/dye solution flows harmlessly through the patient's bloodstream. *— J. Silberner*

some general way ought to look like what we see [in newer crust]. But our observations suggest that's not true."

Either the processes shaping the earth have changed since the planet's early history, or there are some as-yet-undiscovered processes that act on the crust and the upper mantle over very long time periods, changing the crust from the kind of structure scientists see under the Cordillera to that of the craton just to the east of it, says Nelson.

One way to understand the processes that have formed the Moho is to look at many different areas and hope a pattern emerges, he says. "Right now we're just getting a hint of a pattern. But we don't know the reasons for it." — *S. Weisburd*

Alzheimer's disease: Scientists report research advances

Medical scientists cannot tell you the cause of nor administer a cure for Alzheimer's disease, which slowly erodes the minds of 5 to 10 percent of people over 65. But there was excitement and optimism at an Alzheimer's disease research forum held last week in Washington, D.C., at the 16th annual meeting of the Society for Neuroscience. In an unscheduled, last-minute presentation, Dmitry Goldgaber of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) reported the isolation, localization and characterization of a protein and gene possibly associated with the illness. Also notable was the disclosure by two scientists from the Albert Einstein College of Medicine in the Bronx of their discovery of what may be the first accurate diagnostic indicator of Alzheimer's.

In time, these and other research findings reported at the forum could lead to an understanding of the causes of the disease, which affects an estimated 2.5 million elderly people in the United States at an inestimable cost in human suffering and which levies an economic burden soaring into the billions of dollars.

Goldgaber reported results of ongoing experiments that he is conducting in collaboration with Carleton D. Gajdusek, also of NINCDS, and Michael Lerman, Wesley McBride and Umberto Saffiotti, all of the National Cancer Institute. The scientists have identified and localized a gene on chromosome 21 as coding for part of a protein that may be a precursor of amyloid, a small protein that is unusually abundant in the brains of Alzheimer patients. While some scientists have long suspected that the disease is genetically based (SN: 7/2/83, p.5), Goldgaber's research marks the first time a gene has been experimentally associated with the disease.

The researchers have not yet published their results, nor could they have submitted them — the report did not yet exist — before the deadline for this year's Society for Neuroscience meeting. But the importance of their laboratory findings compelled them to circumvent the more orthodox and slower avenues. About a week before the meeting, Goldgaber called symposium chairman John H. Morrison of Scripps Clinic and Research Foundation in La Jolla, Calif., and informed him of his group's results. Morrison agreed that the presentation should be included at the meeting.

Goldgaber emphasized in the news conference that neither amyloid's action in cells nor its abundance in Alzheimer brains is understood. But now that they know the cellular address of the gene coding for amyloid, scientists will be able to compare these genes from Alzheimer patients with those from healthy people, Morrison told SCIENCE NEWS. Any differences in the genes and the gene products would strongly suggest that the gene is at least partly responsible for the devastating disease. Goldgaber says it is too early to say that the gene definitely plays a role in Alzheimer's.

Other research reported in two presentations of the symposium could lead to early diagnosis of the illness, an ability that would be essential for any cure that may be developed in the future. Presently, the only way to make a positive diagnosis of Alzheimer's disease (except in rare cases when brain biopsies are done) is to examine brain cells obtained after the death of the patient. Pathologists look for specific cellular signs that Alzheimer's disease, and not some other disease, was the cause of the patient's symptoms. Such signs include tangles of tiny filaments inside neurons, chaotic assemblages of cellular components and indications that abnormal brain-cell death had occurred before the patients themselves had died.

Peter Davies and Benjamin Wolozin of the Albert Einstein College of Medicine reported their discovery of what might become the first unambiguous early indicator of the disease. Using an antibody probe, the two researchers observed high levels of a protein they call A68 in eight of nine brains from people who had succumbed to Alzheimer's disease. In addition, brain samples obtained from biopsies of two living patients suspected of having Alzheimer's disease had high levels of A68, indicating that the protein accumulates early in the course of the disease.

The scientists developed a technique that allows them to detect A68 in the cerebrospinal fluid of Alzheimer patients. Davies says he hopes the procedure can be used as a diagnostic test, which will greatly facilitate clinical research. In addition to the diagnostic possibilities, the Bronx scientists' work reveals more of the biochemical orchestration that underlies the disease. A68 has "protein-kinase" activity, which makes it like a middle-management protein that regulates the activity of other cellular proteins and enzymes. Also, electron microscope studies show A68 to be associated with neurofilaments in Alzheimer brains.

This evidence, says Wolozin, suggests that A68 may play a role in the the process that leads to the tangles and disorganized plaques observed in certain areas of Alzheimer brains. Scientists suspect that these are signs of neural disorganization that underlie the mental deficits characteristic of the disease.

In other research on diagnostic indicators for Alzheimer's, scientists from the National Institutes of Health and the National Institute on Aging found that Alzheimer patients are not good at discriminating smells. This olfactory impairment, they say, is detectable in "the very early stages of the disease" and therefore might be useful as a signal for the presence of the disease.

Ten other scientists at the forum reported a wide range of findings that describe how brains of Alzheimer victims differ from those of healthy people. A group of scientists from Scripps Clinic and Research Foundation and The University of Psychiatry in Geneva, Switzerland, found that the tangles and plaques seen in Alzheimer brains during autopsies are associated with those cells in the cortex that project long fibers to other cortical cells. Three scientists from the University of British Columbia in Vancouver reported low levels of hippocampal RNA in Alzheimer brains. Other groups from U.S. universities, hospitals and research institutes described possible immunological and viral involvement in the disease, the extent and range of disorganization in neural structures, animal models for studying the illness, and abnormalities in Alzheimer brains of the distribution of proteins, neurotransmitters, hormones and other biological molecules.

These presentations come on the heels of a report of a drug treatment that appears to help alleviate some symptoms of Alzheimer's disease (SN: 11/15/86, p.308).

Although no cure for Alzheimer's disease will precipitate directly from the findings disclosed last week, many medical researchers are optimistic that early diagnosis is in the offing. However, Morrison and other scientists emphasize that they are still very much in the phase of discovering and describing the details of the disease in order to determine exactly what it is they are contending with.

— *I. Amato*

News of the week continued on p. 333