Flight Center in Greenbelt, Md. Instead, he and Ingersoll believe, the sound waves come from Jupiter's tropopause, a region just below the stratosphere and above the ammonia clouds.

Because the temperature rises both above and below the tropopause, sound waves remain trapped in this region, expanding horizontally rather than bending upward from a lower depth, Deming adds. The generation of such waves "is consistent" with the notion that the fragments exploded at or just below the visible cloud tops, he says.

Disappointed that the sound wave didn't originate from a deeper, more intriguing part of Jupiter, Deming notes that features of the wave may still offer important clues about the nature of the Jovian troposphere.

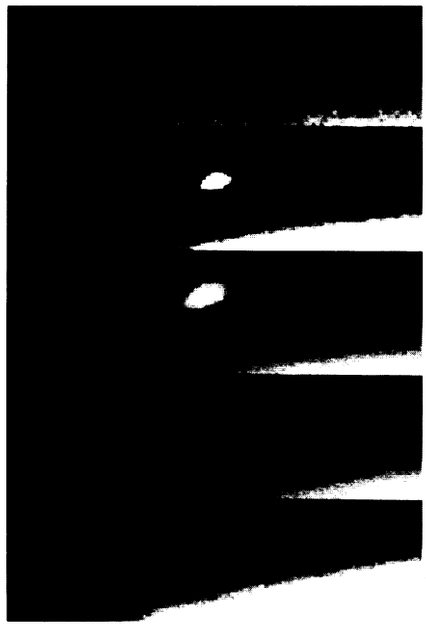
Ingersoll also found evidence of another type of wave nearer the center of the G impact site. He has tentatively identified this fainter, slower-moving dark circle as a gravity wave, whose expanding ripples cause material in Jupiter's upper atmosphere to bob up and down. Ingersoll believes that the slower speed of the gravity wave indicates that it comes from the water-cloud layer, which lies deeper in the atmosphere. At the same time, the faintness of the wave indicates that the G fragment triggered the ripple when it exploded higher in the planet's atmosphere, he says.

Both sound waves and gravity waves produce tiny changes in temperature as they travel through the atmosphere. So why didn't infrared telescopes, which can directly detect such changes, find the waves? Ingersoll proposes that the ripples were so close together that even the highest-resolution infrared instruments couldn't distinguish a hotter-than-average ripple from an adjacent, colder one.

He ascribes their detection in visible light to both Hubble's ability to image small features "and luck." The luck, Ingersoll notes, came because the material surrounding the G impact acted as a visual tracer for the waves, condensings a dark solid around colder ripples and remaining as a relatively transparent gas around adjacent, warmer ones.

Debate continues about whether each fragment consisted of a single solid body or a loosely bound agglomeration of much smaller pieces. The latter model, known as the rubble pile, seemed to fall into disfavor as astronomers witnessed the Jovian fireworks. But Erik Asphaug of NASA's Ames Research Center in Mountain View, Calif., who helped develop the model, responded to critics in a widely circulated electronic-mail message entitled "Rubble Piles Are Not Wimps."

Kevin Zahnle of NASA Ames claims that the model matches the observations and that even the G fragment may have measured no more than 1 kilometer across, one-third the size estimated by Hubble scientists. Images of the actual impacts, taken by the Galileo spacecraft and expected to be radioed in 2 weeks, may help settle part of the controversy. — *R. Cowen*



STSI/NASA

On July 18, Hubble captured this sequence showing the emergence of a plume and its flattening as it sinks back into the Jovian atmosphere.

Does a virus cause some kids' asthma?

For some children with asthma, no amount of medication seems to relieve their wheezing. New research now suggests that certain children with hard-to-treat asthma may suffer from a smoldering viral infection of the lungs.

Asthma specialists have always known that some children develop the disorder after suffering an acute bronchial infection with a bug called adenovirus. Scientific dogma holds that the body's immune system clears this virus, but an infection can leave the lungs vulnerable to a chronic condition in which pollutants in the air and other allergens trigger breathlessness.

Vasilija Maček of the University Medical Center in Ljubljana, Slovenia, and her colleagues wondered if such children continue to wheeze because they never really got rid of a lung infection. To test that hypothesis, they recruited 34 children who had recovered from such an acute infection and had then been diagnosed with asthma. All 34 had failed to breathe more easily with conventional treatments, such as steroid drugs and bronchodilator medication.

The team began its study by inserting a slender tube into each recruit's trachea and collecting fluid samples from their lungs. The researchers discovered a protein made by the adenovirus in samples from 31 of the 34 children. When the researchers tested 20 children who did not suffer from asthma, they found no sign of this viral protein. Such evidence is suggestive but does not prove that the virus is actively replicating in the lungs, comments Hugh O'Brodovich, a lung specialist at the Hospital for Sick Children in Toronto.

Next, the team took lung-fluid samples from 6 of the 31 children and successfully grew cultures of adenovirus from them. "I think it's a very provocative finding," O'Brodovich says. That evidence indicates that the virus is actively replicating in the lungs of these children, he adds.

The Slovenian investigators describe their findings in the July AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, a journal published by the American Lung Association. The results suggest that some kids fail to quash an adenovirus infection, continuing instead with a low-grade infection that leads to chronic breathing problems. Still, these findings have yet to be confirmed, O'Brodovich says, noting that this is the first study to implicate adenovirus directly in the development of asthma.

The new research hints that antiviral therapy might benefit certain kids with intractable asthma, adds Maček. She hopes her findings will spur drug developers and clinical researchers to take a hard look at the impact of antivirals on such cases.

Does a viral infection underlie other cases of asthma, including some in the adult population? Maček suspects so, but she says further study must answer that question.

There's evidence that adenovirus also plays a part in a different respiratory ailment. James C. Hogg of St. Paul's Hospital in Vancouver, British Columbia, and his coworkers have found previously that some smokers who develop chronic obstructive lung disease show evidence of adenovirus infection.

Hogg, who wrote an editorial to accompany the Solvenian report, also points out that the new results raise a red flag for the emerging field of gene therapy. Genetic researchers have employed a crippled adenovirus to carry a therapeutic gene into the pulmonary cells of cystic fibrosis patients. Maček and her team point out that some people may have a latent adenovirus infection. Hogg worries that the crippled virus may combine with the virus already in the lungs and start to spread.

— *K.A. Fackelmann*