

receptors on the vessels were abnormal. Such receptors, which control the expansion and contraction of cerebral blood vessels, are known not only to receive signals from incoming involuntary nerves but to be influenced by CO₂ and other chemicals in the blood.

Meyer and his team decided to test whether these receptors were responsible. They gave migraine patients drugs that are known to be both clinically useful for countering migraine pain and capable of stimulating alpha and beta nerve receptors on cerebral blood vessels. They then measured the patients' cerebral blood flow response to CO₂. Their results, reported at this year's AASH meeting, showed that this group of drugs tended to lessen the overreaction to CO₂ of the migraine patients. This finding, Meyer and his co-workers conclude, implies that the excessive responsiveness of migraine patients' cerebral blood vessels is due, at least in part, to abnormal alpha and beta receptors on the vessels.

In spite of these discoveries, the method by which abnormal cerebral blood flow leads to head pain during a migraine is not clear. It is possible that when cerebral blood vessels overreact to nerve messages or other chemical stimuli they first constrict quickly, subsequently expanding excessively. This could account for the initial cerebral blood flow decrease and the increased flow during the headache phase.

Such an excessive expansion might impinge on pain-carrying nerves, which then send pain messages to pain centers in the brain.

What is now known for sure about migraine pain is that enkephalins become depleted at the beginning of the migraine attack but increase to normal levels as the headache subsides (SN: 6/21/80, p. 390). This finding, part of the research of Bruno Anselmi and co-workers at the University of Florence in Florence, Italy, was given the 1980 Harold G. Wolff Award at the AASH meeting and, along with the research of Meyer and colleagues, provides an indication that the researchers are on the right track: "What we found and what Anselmi found are complementary," Meyer told SCIENCE NEWS.

Taken all together these findings suggest that migraine sufferers have abnormal nerve receptors on cerebral blood vessels — abnormal receptors that allow the vessels to react excessively to gases, nerve messages or other stimuli and thereby bring about abnormal cerebral blood flow, usually a decrease, then an increase. The stretched vessels stimulate nerve fibers for pain. And thus when cerebral blood flow is increased, it results in pain stimulation that seems to cause, or at least be accompanied by, a depletion of brain pain-relieving chemicals. The results: excruciating head pain known as a migraine. □

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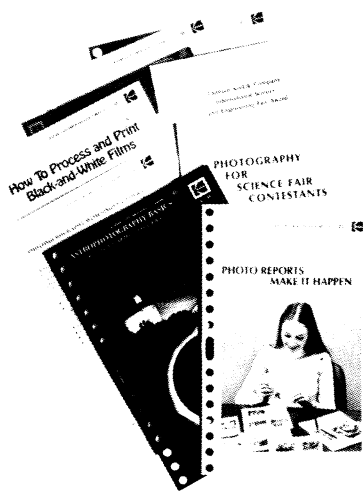
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