heard, to estimate their distance from shore. With an atomic clock, a submarine navigator can compare "his time" to that broadcast by a radio transmitter on shore and, using the speed of light to calculate the delay, arrive at his position. A two-microsecond delay could thus make a life or death difference—an error in position of about a third of a mile.

At least man-made clocks can keep better time than the earth: To compensate for slowing of the earth's rotation, both the French and American clocks are momentarily stopped about once a year for a "leap second." Since 1958, when atomic clocks were first used to keep track of such matters, the earth has slowed nearly 14 seconds. □

Vitamin C: Again on the defensive

It's hardly surprising, when Americans suffer from 90 million colds annually, that they'll grab at anything that might spare them their misery. And the preventative that is much sought after these days, thanks largely to Nobelist Linus Pauling, is vitamin C.

Many people who take vitamin C regularly to ward off colds swear by it. So do a number of scientists. Pharmacologists Erwin D. Cyan writes in his book *Vitamins in Your Life*: "It is our forecast that the use of vitamin C in the common cold will be accepted in the future. In some studies, such as the Anderson/Toronto one, it seems already to have been vindicated."

Other scientists, however, remain unconvinced by the Anderson study and other research results that support vitamin C's role as a cold preventative. Examples are Michael H. M. Dykes, senior scientist at the American Medical Association Department of Drugs; Paul Meier, a pharmacologist at the University of Chicago, and Thomas R. Karlowski and his team at the National Institutes of Health. They report their criticisms of past studies and new negative research findings in the March 10 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.

Dykes and Meier analyzed more than a dozen studies conducted on vitamin C during the past 35 years, including the Anderson study in Toronto in 1971-72. This study found that 26 percent of 407 subjects who took one gram of vitamin C daily to ward off colds were spared colds, compared with 18 percent of 411 control subjects who took placebos. The study, Dykes and Meier admit, "was double-blind and appears to have been well controlled and not subject to many of the criticisms applicable to the other discussed here." However, Anderson and his team conducted another study in 1973, to get a clearer idea of vitamin C's value as a cold preventative. "In this study," Dykes and Meier point out, "all the differences between measures of illness were small compared to the standard errors, and none approached statistical significance."

Thus Dykes and Meier conclude in JAMA: "Although one study tentatively supports the hypothesis that such doses of ascorbic acid may be efficacious, a second study by the same group did not confirm the significant findings, and no clear, reproducible pattern of efficacy has emerged from the view of all the evidence."

In a new study, Karlowski and his team had 311 employees at the NIH take three grams of vitamin C or a placebo daily for nine months. This would be equivalent to the amount of vitamin C that one would get eating 100 oranges daily. One hundred ninety volunteers completed the study. Those getting vitamin C as a preventative had 1.27 colds in nine months; those receiving a placebo, 1.36 colds. If volunteers felt they were coming down with a cold, they were given an additional 3 grams of either vitamin C or placebo daily. Those who received their usual placebo plus an extra placebo had colds for an average duration of 7.14 days. Those who received their usual three grams of vitamin C plus a placebo had colds for an average duration of 6.59 days. Those who received their usual three grams of vitamin C plus an extra three grams of vitamin C had colds for an average of 5.92 days. "Analysis of these data," the investigators conclude in JAMA, showed that vitamin C "had at best only a minor influence on the duration and severity of colds." □

New insight into dystrophic muscle

The hallmark of muscular dystrophy, specifically the Duchenne variety, is that it strikes the very young. A victim continually falls and has trouble getting up as he takes his first uncertain steps in life. By the time he reaches adolescence, his muscles are so weak that he must stay in a wheelchair. Chances are slim that he will live to see his 21st birthday. There is no treatment that can save him.

Obviously the way to conquer this tragic disease is to get at the cause of it. It is known to be inherited. So the cause probably lies in some genetically expressed metabolic defect. But what kind of a defect? Evidence that the defect lies in nerves that innervate skeletal muscles is equivocal at best, W. G. Bradley pointed out in NATURE last summer (250: 285). Several investigators reported that 27 percent of skeletal muscle fibers in mice with muscular dystrophy were denervated. But other scientists, found that motor nerves in patients with Duchenne muscular dystrophy are quite capable of sprouting to reinnervate denerved muscle fibers and thus show no sign of being sick.

Evidence now reported in NATURE (253:464) strongly suggests that the metabolic defect underlying muscular dystrophy is a faulty sugar-metabolizing enzyme. The evidence comes from Jennifer M. Strickland and David A. Ellis of the Midland Centre for Neurosurgery and Neurology, Warley, Britain.

Strickland and Ellis first found, in dystrophic muscle, that more glucose than usual was converted to fructose instead of to glucose-6-phosphate. The conversion of glucose to glucose-6-phosphate is catalyzed by an enzyme called hexokinase. Thus it appeared that hexokinase was not working properly in muscular dystrophy. So the investigators set up tests to see whether this was the case.

They studied both the amounts and movements of hexokinase isoenzymes (varying molecular configurations of hexokinase) in various muscle samples. The samples included healthy muscle, polio-afflicted muscle, muscle with motor neuron disease, muscle with Duchenne muscular dystrophy, muscle with a so-called Becker muscular dystrophy, and so on. They found that the isoenzymes did not vary significantly in amount from one muscle sample to another. This finding confirmed what other investigators have found—that hexokinase appears to be present in adequate amounts in dystrophic muscle. But Strickland and Ellis did show that one of the isoenzymes—known as isoenzyme II—varied in movement in two of their muscle samples. The samples were muscle with Duchenne muscular dystrophy and with Becker muscular dystrophy. "As this isoenzyme is characteristically abundant in skeletal muscle," the investigators assert, "the change in its properties in dystrophy may be very significant."

They also found the same aberrant movement in isoenzyme II in three livers, one brain and one sciatic nerve obtained after death from patients with Duchenne dystrophy, and from muscle and liver from a fetus of 18 weeks diagnosed with Duchenne dystrophy.

So they conclude that defective hexokinase enzyme, specifically in its variant known as isoenzyme II, may constitute the basic metabolic defect underlying muscle dystrophy. And since the isoenzyme is also present in nerves and liver, its abnormal behavior may explain why muscular dystrophy often damages not only muscle but these other tissues. □