unconsciousness and lack of breathing. The child became increasingly clumsy, suffered mental deterioration and died before his second birthday.

While the child was alive, Brandt and colleagues worked hard to diagnose the disease and to treat it. What struck them most was that the symptoms mimicked those of acute morphine poisoning. Thus they suggested that the disease might be caused, at least in part, by an excess of enkephalins — the two endorphins that seem to be the brain's own natural morphine or pain-killing molecules. The researchers took samples of spinal fluid from the child and found an excess of endorphins (most likely enkephalins) — about 100 times greater than that found in healthy children of the same age. Then the researchers gave the child naloxone, which is known to counter the effects of both morphine and the enkephalins, in hopes that it would help. The first three times the naloxone had no effect; the fourth time it did. Within several minutes after naloxone injection, the child emerged from his coma and embraced his mother. Unfortunately, further naloxone injections didn't help. Nonetheless, the fact that naloxone had produced at least some improvement in the child is further evidence that an excess of enkephalins could in some way be involved in the disease. Another sign that this might be the case was the child's unusual resistance to pain.

After death, autopsy provided still more evidence linking the disease with an excess of enkephalins. Tissue from the child's cortical and subcortical brain areas was found to contain enkephalin levels 100 or more times greater than those of control children. In contrast, levels of beta-endorphin (another endorphin) were barely detectable in the victim's cortical and subcortical brain areas and did not differ in levels from those of control children. Thus, excessive enkephalin levels probably caused, or at least contributed to, the child's disease, Brandt and colleagues conclude. And in his editorial, Snyder agrees: "Presumably, the patient's gross brain-stem degeneration had elicited clinical manifestations, including coma, through release of excess enkephalin. Such a profound role for the enkephalins in regulating brain function has never before been proposed. The extraordinary changes in brain levels of enkephalin, however, would fit such dramatic conclusions."

Snyder points out, though, that weaknesses in the study techniques Brandt and his colleagues used might have erroneously led to the above results. And Brandt and his colleagues admit that they are not sure if excessive enkephalin levels were the initial cause of the child's disease or a secondary one, because the child's brain gave evidence not only of elevated enkephalin levels but of a genetic disease called Leigh's syndrome. □

Alligator rhythm without a pineal

Alligators move out of the water in the morning and back into the water at night. They breed in the spring and become less active in the fall. Such daily and seasonal rhythms are sustained even though alligators have no pineal gland, the organ generally thought to regulate temperature, activity and reproductive rhythms through its cyclical production of the hormone melatonin (SN: 10/11/80, p. 229).

Experiments with alligators are difficult because their large size makes them hard to manage, says Jan J. Roth of the National Institute of Mental Health. He and colleagues, however, succeeded in sampling the blood every three hours of five alligators at the St. Augustine (Fla.) Alligator Farm. They found a constant low level of melatonin, similar to that of animals whose pineal glands have been surgically removed. This melatonin may come from a gland in the corner of the eye, the retina or intestinal cells. But Roth says it probably does not regulate the daily cycles of alligator life.

Alligators are the modern representatives of the subclass of reptiles to which dinosaurs belong, and fossil skulls indicate that most dinosaurs, like alligators, had no pineal gland. Roth and colleagues suggest in the Oct. 31 SCIENCE that the dinosaurs evolved during a period of less dramatic seasonal change. In a continually warm climate, less precise timing of daily and seasonal activities may be sufficient, Roth explains. But with the glaciers of the Ice Age, animals again needed to be keenly sensitive to differences in seasonal conditions, and only a few animals lacking pineal glands survived to modern times. Today alligators inhabit only warm climates, as do anteaters, armadillos, sloths and manatees, the other extant animals lacking pineal glands. □

The effects of stress at the cellular level

Stress during pregnancy can affect one's offspring, some animal studies and clinical observations have suggested. Among the possible results are cleft palate, harelip, aberrant sexual behavior, irritability, hyperactivity and feeding problems. Now stress during gestation has been found to exert an even more serious effect, according to a study reported in the Oct. 31 SCIENCE by G. Miller Jonakait, Martha C. Bohn and Ira B. Black of Cornell University Medical College in New York City. Their work with rats suggests that stress can alter the way embryonic nerve cells express their genetic potential. "That is a change at a very fundamental level," Black told SCIENCE NEWS.

Jonakait and her colleagues stressed pregnant rats pharmacologically with reserpine, an antipsychotic drug that, when given to pregnant animals in the past, has resulted in neurological or behavioral abnormalities in their offspring. But this time the researchers were attempting to see what effect reserpine had, if any, on a select population of developing central nerves in fetal rats. They found that reserpine extends the period during which the cells normally express the neurotransmitter noradrenaline. Thus reserpine was found to be capable of altering the phenotypic expression of developing embryonic cells (that is, of altering the expression of their genetic potential). Presumably stressors besides reserpine could produce still other effects of this nature, the researchers believe. For this reason, along with the connection already made between prenatal stress and birth defects, they counsel pregnant women to avoid stress as much as possible during pregnancy.

The researchers next attempted to determine how reserpine exerted its effect on embryonic nerve expression. Because reserpine is known to increase blood levels of glucocorticoids, they suspected that these adrenal steroid hormones might be a link between reserpine and the alteration of embryonic nerve expression. They found that this was the case. For instance, when they implanted glucocorticoids in pregnant rats, the hormones produced the same effect on the developing nerves as did reserpine: The nerves expressed noradrenaline beyond their usual time. And when a glucocorticoid inhibitor was injected into pregnant rats, reserpine did not extend the production of noradrenaline in the embryonic nerve cells. This is "the first time that maternal glucocorticoids have been found to directly affect phenotypic expression of neurons in the fetus," Jonakait explains. Still other maternal hormones besides the glucocorticoids may mediate other stressful effects on fetal nerve expression, she, Bohn and Black contend. In other words, they may have just uncovered the tip of an iceberg as far as the ability of stress to alter embryo phenotypic expression goes and as far as the intermediary role that hormones may play in such alterations is concerned. □