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with Jack Ryan

be able to reduce the chances of cellular growth getting off track during a baby's development.

Consider cleft palate again. Two New Jersey doctors, Lyon P. Streen and Lyndon A. Peer, made a study of mothers of cleft-palate children. Most had suffered some illness or emotional disturbance, and in each case at a stage of pregnancy when the palate forms. Could these stresses have caused the mothers' adrenal gland to produce extra hydrocortisone which, in turn, retarded normal formation of the baby's palate?

The doctors injected female mice with cortisone and other agents in heavy dosage. The mice produced offspring with cleft palates. In the next experiment, the same agents were introduced into the female mice—but while the offsprings' palates were forming, the mice also received vitamins B6, B12, and C. This time, no cleft palates. Had the vitamins countered the original agents? If so, would it be practical to give these vitamins to an expectant mother who suffers illness at a stage of pregnancy when the palate forms?

No preventive has come out of this experiment, but it shows the direction research is taking in trying to learn what causes abnormalities and how to reduce them.

This is important because we are not sure how permanent corrective surgery is. The cleft-palate child probably will have no future trouble. But what of the baby whose heart did not develop properly?

UNTIL 20 years ago, he faced death or incapacitation. Then Dr. Robert Gross performed an operation on an infant's heart, closing off a malformed duct that was shunting blood from its normal circulatory route. From that pioneering effort, surgeons have advanced until they now probe the heart itself, mending nature's mistakes while machines take over the job of circulating blood.

But if the heart had developed correctly, there would be less question of its enduring in adolescence and adulthood. Was this an inherited defect? Or did some agent thwart nature—a drug the mother took, something awry in her body chemistry, or a virus infection?

In 60 percent of the cases, we don't know what caused the anomalies, although scientists believe most congenital defects result from a combination

of causes. Are parents largely to blame for the defects? Sometimes they think so, yet there is little scientific basis for this feeling. Only 20 percent of abnormalities have been traced to inherited factors. Ten percent probably come from chromosome aberrations (a breakdown of the chromosome, which helps pass on parents' characteristics, by nongenetic causes—radiation would be one example), and another 10 percent from virus infection.

THE THALIDOMIDE tragedies illustrate how more than one factor is probably involved in a deformed birth. You recall headlines about deformed babies being born to many mothers who took the drug, but what we overlooked is that deformities occurred only among 20 percent of thalidomide mothers. Why did most of them bear normal children? One guess is that the drug alone did not cause the deformity. It was a combination of the drug and other factors.

While we grope toward those "other factors," initial research has provided young women with a guide for reducing the risk of bearing deformed children. Abnormalities develop in the first three months of pregnancy, with the first six weeks most vital, so obviously a woman should be under a doctor's care as early in pregnancy as possible.

She will be advised to avoid such anomaly risks as X rays of the pelvic region, certain drugs, and high altitudes. Some doctors may suggest immunization against influenza because we know that at least one virus can adversely influence development of the embryo and may lead to abortion. In 1941, Dr. N. M. Gregg of Australia first proved that German measles, contracted during the first three months of pregnancy, produced heart malformations, cataracts, deafness, and other defects in the child.

Despite such evidence, doctors could do little but warn expectant mothers of the risk of infection. Perhaps they will do more in the future. Just last year American scientists succeeded in isolating the elusive virus that causes German measles. Evidence indicates that 90 percent of those infected develop neutralizing antibodies to the virus. There is a tremendous amount of work to be done on this development, but some hope is offered for a vaccine which

would immunize child-bearing women against German measles.

Are there other anomaly-producing viruses? Last year Australian doctors found indications that mongolism, which results from the presence of an extra chromosome, may be caused by a virus. For 16 years they kept records of mongoloid births, and the pattern typified those associated with viral epidemics. Mongoloid births clustered more in urban areas where there is more personal contact, and they increased in number at certain times—one Melbourne suburb averaged one or two mongoloids a year, then in 1944 seven were born in seven months, four in the same week and living within a quarter-mile area. Fifteen years later, five mongoloids were born in the same suburb within a half-block area. This is no more than a beginning, of course, but if borne out by other studies, we could hope to isolate the harmful virus someday, then try to develop an "antimongoloid vaccine."

MEANWHILE, other scientists concentrate on helping those already born with afflictions. Significant successes, for example, have been achieved with metabolic disorders. A Swedish mother with two healthy children loses her newborn, and nobody knows why. But she recalls an uncommon odor to the baby's diapers, and from that slim clue eventually evolves treatment for phenylketonuria (PKU).

In PKU, the liver fails to produce an enzyme which acts on phenylalanine. When unconverted, this life-building amino acid gets into the blood stream with poisonous effects. That clue in Sweden led to the first forward step, a "green diaper" test which alerted doctors to the defect. Next came the development of a diet low in phenylalanine which saved the afflicted baby's life but, because the diaper test lost vital time, it was sometimes administered too late to prevent serious brain damage.

Obviously, the next step was to find a quicker test. One has recently been developed. It uses a few drops of blood from the baby's heel to diagnose the disorder. Last summer, the U.S. Children's Bureau began a screening of this test on 400,000 newborns, with expectations that it will permit earlier treatment of PKU and other metabolic disorders.

Among the metabolic disorders

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