In 1965 Dr. Nora and his associates had noted that 3 mothers of children born with the congenital heart defect of transposition of the great vessels had taken an anti-obesity drug, dexamphetamine sulphate, during the early weeks of pregnancy. They then determined whether dexamphetamine sulphate could produce in pregnant mice congenital malformations especially congenital heart defects. They administered the drug in approximately 20 to 50 times the human dose to the mice in order to maximize the chances of detecting malformations. They found that in addition to cleft lip and eye abnormalities cardiac malformations were produced in the experimental animals and not in the controls. Thus, a single very large dose of dexamphetamine sulphate can produce congenital cardiac and other malformations in mice. Moreover, another appetite suppressant, phenmetrazine, has also been shown to produce congenital defects in animals. In 1967 Dr. Nora's group in a retrospective analysis of 219 cases and a prospective study of 52 cases involving use of dexamphetamine sulphate during human pregnancy yielded no teratogenic effects. The question was reconsidered in 1970 however, when a subsequent study of 184 mothers of infants with heart malformations showed a higher incidence of amphetamine ingestion than a controlled group. Another study has found an elevated incidence of biliary tract atresia among offspring of mothers taking amphetamines. Some confirmation of these suspicions comes from the retrospective survey of Scottish mothers which showed a higher proportion of children with various malformations including congenital heart defects from mothers who had taken dexamphetamine for suppression of appetite during the early part of pregnancy than the control mothers of normal infants. Two studies also show that phenmetrazine was associated with