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The Prevalence Rate of Congenital Heart Disease in Newborn Gerbils (Meriones Unguiculatus)

A Preliminary Report

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A preliminary report on the prevalence rate of congenital heart disease in newborn gerbils (Meriones unguiculatus) is presented. Serial sections of the entire heart of 100 liveborn gerbils were studied. The prevalence rate of congenital heart disease was 4%. Ventricular septal defect was the most frequent lesion. This is in agreement with previous reports on rats.

The experimental production of congenital heart disease (CHD) in animals may shed light on its etiology and prevention in man. This paper is a preliminary report on the incidence of CHD in newborn gerbils (Meriones unguiculatus).

Materials and Method

Newborn gerbils were anesthetized by ether and sacrificed before their seventh day of life. After their entire thoraces were fixed in 10% formalin solution, the heart-lung complexes were removed and processed in 50%, 95% and 100% alcohol and subsequently cleared by xylene. The heart-lung preparations were then embedded in paraffin and sectioned serially. The sections were stained by hematoxylin-eosin and studied under a low power microscope (25 times magnification).

No dead newborns were included. All litter mates were examined. The mothers of the newborns were apparently normal and they received no drugs prior to, during or after conception.

Results

Size of the litters ranged from three to eight animals in each litter (mean 4.7). The litter size of the affected animals was no different from the normal ones. The animals were examined before sacrificing and no gross abnor-
malities, such as cleft lip or defects of ears, extremities or tail were present.

Four instances of ventricular septal defect (VSD) were detected, an incidence of 4%.

The defect in specimen #23 was a rather large muscular VSD located in the posterosuperior portion of the ventricular septum leaving the posterior portion of the medial cusp of the tricuspid valve unprotected (Fig 1). This lesion was accompanied by a very dilated right atrium as well as a functionally patent foramen ovale (Fig 2). The structure of the interatrial septum was normal; therefore, the incompetence of the valve mechanism was probably secondary to dilatation of the atrium resulting from VSD. Specimen #33 had a muscular VSD in the upper portion of the interventricular septum that appeared to be functionally significant. This defect was also accompanied by a functionally patent

Figure 1
Specimen #23 with a muscular VSD. Note the dilated right atrium. Ao: aorta; LA: left atrium; PA: pulmonary artery; RA: right atrium; LV: left ventricle; RV: right ventricle; TV: tricuspid valve.

Figure 2
Specimen #23 with functionally patent foramen ovale. FO: foramen ovale; LA: left atrium; LV: left ventricle; MV: mitral valve; RA: right atrium; SVC: superior vena cava.
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foramen ovale and an incompetent valve mechanism (Fig 3). The defect in specimen #32 was in the form of a very small membranous VSD connecting the left ventricle at the root of the aorta with the right ventricle just under the medial cusp of the tricuspid valve (Fig 4). Specimen #39 had a membranous VSD in the form of a very narrow tortuous canal undermining the septal cusp of the tricuspid valve thus producing a communication between the left and the right ventricles. The diameter of the defect in this case was approximately 0.15 mm (Fig 5 & 6). The significance of this lesion and its pathogenesis is the subject of a separate report.* Specimens #33 and #39 were of the same litter.

The defects in cases #32 and #39 were functionally insignificant. There was no case in this series of patent ductus arteriosus nor any degree of discernible pulmonary outflow tract.

* Published this issue.

Figure 3
Specimen #33 with muscular VSD. Ao: aorta; LA: left atrium; LV: left ventricle; PA: pulmonary artery; RA: right atrium; PV: right ventricle.

Figure 4
Specimen #32 with small VSD. Ao: aorta; LA: left atrium; LV: left ventricle; PA: pulmonary artery; RA: right atrium; RV: right ventricle; TV: tricuspid valve; VS: ventricular septum.
obstruction or right ventricular hypertrophy. No heart abnormalities were found in a second litter born to the parents who produced defective specimens #33 and #39. Two litters whose parents were first degree relatives of these parents were also normal.

Discussion:
In a review of the epidemiology of the CHD, Higgins\textsuperscript{1} states that the information obtained by experimental production of heart disease in laboratory animals may throw light on CHD in man. The first step towards such experimental production in gerbils is to establish the types and incidence of CHD in the normal animal population.

The hearts of 100 newborn gerbils from 21 litters were studied by serial section technique. The overall incidence of CHD in this series was 4%. The only defects found in these animals involved either the membranous or the muscular portion of the ventricular septum. The former lesion was functionally insignificant. In two specimens a large muscular VSD was accompanied by a very dilated right atrium and functionally incompetent valve of the foramen ovale.

The incidence of CHD in man is estimated to be approximately 0.5%\textsuperscript{1} of all births with reports varying from 3-9/1000. There are few reported studies on the incidence of CHD in animals. A screening study by Detweiler et al\textsuperscript{2} on 1,000 dogs of varying ages revealed four cases of CHD and several cases of acquired heart disease. The diagnoses of pulmonary stenosis in three animals and patent ductus arteriosus in one were confirmed by autopsy. Higgins\textsuperscript{1} interprets a frequency of 4/1000 as an indication that the prevalence of CHD in dogs is not very different from that in man. However, this may not be true because the four cases of CHD in Detweiler's series were found in dogs 0-2 years old and the incidence of CHD in this age group was 7.8/1000.

In 1961 Detweiler\textsuperscript{3} and his associates reported spontaneously occurring heart disease, acquired as well as congenital, in 3,000 dogs with 15 cases of definite CHD in addition to acquired valvular and coronary disease. Thus, the frequency of all congenital heart defects was 5/1000 in this series. The abnormalities found were pulmonary stenosis (6 instances); patent ductus arteriosus (4); aortic stenosis (3); ventricular septal defect (1) and tetralogy of Fallot (1). In 337 instances of clinical heart disease in the 3000 dogs, a definite diagnosis could not be established. One can assume that the incidence of CHD could have been higher than 5/1000 if definite diagnosis had been made in all dogs.

The incidence of CHD in rats has been reported to vary from 3.2% to 7.5%. Experimenting on the effects of prenatal hypoxia in rats, Haring\textsuperscript{4} found an incidence of 3.2% of CHD (mostly a slit in the membranous portion of the interventricular septum) in 124 control animals. In later experiments\textsuperscript{5} on the role of hypercapnia and hypoxia as etiological factors, she reported an incidence of 4.5% CHD in her control group consisting of 111 rats. Although the most frequent lesion was a VSD, there were also cases of narrowing of the pulmonary outflow tract and patent ductus arteriosus.

The highest incidence of CHD in rats (7.5%) was reported by Clem-
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Figure 5
Specimen #39 demonstrating a small tortuous VSD undermining the septal cusp of the tricuspid valve. Ao: aorta; LA: left atrium; LCTV: lateral cusp of the tricuspid valve; LV: left ventricle; MCTV: medial cusp of the tricuspid valve; MV: mitral valve; PA: pulmonary artery; RA: right atrium; RV: right ventricle. The area within the dotted square is magnified in Figure 6.

Figure 6
Higher magnification of the area within the dotted square in Figure 5 demonstrates the canal-like VSD under the medial cusp of the tricuspid valve. Because of its tortuosity, the full extent of the defect cannot be seen on one single section.
mer et al, who found three instances of small VSD in 44 animals. The present study, based on a small number of gerbils, agrees with the reports on rats, both in terms of incidence as well as the types of CHD.

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