tures going *out* of focus over the same range succeeded in 76 percent of the cases—a highly reliable difference.

Do individual subjects differ in their ability to recognize pictures? Kendall's measure of concordance, W, was used to test the consistency of recognition scores of the 13 standardizing subjects. The result was not significant ($W = .116$, $p > .50$), suggesting that there is no general recognition ability under these experimental conditions.

In summary, exposure to a substandard visual display has the effect of interfering with its subsequent recognition. The longer the exposure and the worse the display, the greater the effect. Examination of the responses of the standardizing subjects, who reported aloud from the start of each picture, provides a clue as to the nature of the interference effect. Hypotheses about the identity of the picture are made despite the blur. The ambiguity of the stimulus is such that no obvious contradiction appears for a time, and the initial interpretation is maintained, even when the subject is doubtful of its correctness.

An incorrect interpretation of the picture may occur either in the primary figural organization of the picture (for example, an inhomogeneity is seen as concave, whereas it is convex in the full picture when correctly identified), or in the assignment of identity to a visual organization (the convexity is recognized, but is seen as a pile of earth rather than correctly, say, as a dish of chocolate ice cream). The amount of exposure necessary to invalidate an incorrect interpretation seems to exceed that required to set up a first interpretation, so that at any particular clarity of the display, those who see it for the first time are more likely to recognize the objects than those who started viewing at a less clear stage.

When one views a picture going *out* of focus, both initial clarity and resistance to change of interpretation are pitted in favor of correct recognition, which accounts for the great superiority of this condition. Indeed, it is striking how long one can "hang on" to the identity of a picture which is going out of focus, considering the difficulty of recognizing the same picture when it is seen for the first time coming into focus.

JEROME S. BRUNER
MARY C. POTTER
Center for Cognitive Studies, Harvard University, Cambridge, Massachusetts

References and Notes

1. D. Galloway, unpublished thesis, University of California, Berkeley (1946); D. Wyatt and D. Campbell, *J. Abnormal Soc. Psychol.* **46**, 496 (1951); P. Gump, unpublished thesis, University of Colorado (1955); A. Crowell, unpublished thesis, McGill University (1961).
2. Since 13 subjects were used in the standardizing group, the point at which the fourth subject recognized the object was taken as the "first quartile."
3. Since there were unequal numbers of subjects in the various conditions, a method of approximation described by Walker and Lev (*4*) was used.
4. H. Walker and J. Lev, *Statistical Inference* (Holt, New York, 1953), pp. 381–382.

6 March 1964

Myocardial Infarction: A Response to Social Interaction among Chickens

Abstract. *A study of male and female chickens caged alone, in male-female pairs, in groups of four males and in heterosexual groups of 6, 12, and 24, with a proportion of two males to one female, suggests that coronary arterial disease with myocardial infarction may be a response to social interaction, especially interaction that relates to sexual behavior. Myocardial infarcts were limited to males of heterosexual pairs and groups and to females of groups that contained 24 chickens. The lesion was found in males that died at 16 to 44 weeks of age, and in females at 32 to 43 weeks.*

Coronary arterial disease with myocardial infarction was found in about 15 percent of mammals and 10 percent of birds that died in the Philadelphia Zoological Garden from 1 January 1954 to 31 December 1963. These values represent an increased frequency of about tenfold for mammals and fivefold for birds over corresponding records of 1944–1953. Earlier encounters with this disease complex were extremely rare.

This animal collection has been fed the same high-quality diets since 1935, about 20 years before the incidence of myocardial infarction began to increase. However, a life-span of 20 years has more often been a maximum than a mean for many types of animals. Thus, while improved nutrition has increased longevity, neither age nor diet may be related to the increased frequency of myocardial infarction (*1*).

Instead, increased frequency of this disease complex has followed an attempt to establish and maintain breeding pairs and breeding groups of many species of mammals and birds. This departure from earlier practices has also set in motion complex behavioral responses (social interactions) inherent to maturation and reproduction, especially of captive wild animals. Apparently, then, frequency of myocardial infarction in this animal collection reflects intensity of social interactions (*1*).

Tests of this assumption with chickens demonstrated that intensity of social interactions, measured directly by frequency of conflict and indirectly by weights of adrenals and gonads, is a function of group size (groups of 6 as opposed to groups of 12). Further, these chickens developed advanced grades of coronary arterial disease within 35 weeks (*1*, *2*). Hence, in our experiment heterosexual groups of 6, 12, and 24 (with a proportion of 2 males to 1 female) were compared with groups of 4 males each, with male-female pairs, and with males and females caged alone. This is a report of the mortality pattern and the occurrence of myocardial infarction that resulted under these conditions.

Birds from a closed, pullorum-free flock of single-comb Hy-line White Leghorns were hatched in one lot, brooded as one flock until they were 7 weeks of age, and then assigned randomly to cages in one large room with 15 hours of light per day. The birds were vaccinated and the sharp edges removed from their beaks at appropriate intervals. Cage assignments and number in each were as follows: 32 males caged alone; 32 females caged alone; 32 male-female pairs; 32 males, 4 to a cage; and heterosexual groups (2 males to 1 female), 12 groups of 6, 10 of 12, and 8 of 24 (total, 544 birds).

Two-tiered commercial battery cages were divided by wooden partitions to allow 2 ft^2 (0.186 m^2) per bird. Continuous food troughs were attached to the cage fronts, and automatic water cups assured continuous access to food and water. Commercial rations were fed. Water was pumped from a deep well. Sexual maturity occurred between 18 and 21 weeks of age, when males weighed about 5.5 lb (2.5 kg) and females about 3.5 lb (1.5 kg).

The experiment was ended during week 45 when survivors were killed for study. Birds that died were examined within 10 hours of death for abnormalities of hearts, livers, spleens, kidneys, gonads, adrenals, and gastro-intestinal

Table 1. Experimental design, and percentages of deaths from all causes to 45 weeks of age and of deaths associated with myocardial infarcts.

Cage population	Total number		Deaths (%)			
			All causes		With infarcts	
	Males	Females	Males	Females	Males	Females
1 male	32		6.3		0.0	
1 female		32		0.0		0.0
1 male and 1 female	32	32	25.0	6.3	9.4	0.0
4 males	32		9.4	0.0		
4 males and two females	48	24	27.1	12.5	4.2	0.0
8 males and 4 females	80	40	21.3	32.5	7.5	0.0
16 males and 8 females	128	64	37.5	37.5	7.8	4.7
Totals	352	192	25.6	21.8	5.9	1.6

tracts. Blocks of tissue from these organs were fixed in 10-percent neutral formalin, embedded in paraffin, sectioned at approximately 5 μ and stained with hematoxylin and eosin. Hearts also were stained for elastic fibers and fibrous tissues, and for evidence of muscle necrosis by the cresyl violet-acid fuchsin-orange G method, and by Lillie's allochrome method. More advanced muscle necrosis was recognizable without special stains and was sometimes accompanied by leucocytic infiltration.

Deaths from all causes and deaths associated with myocardial infarction are shown in Table 1 as percentages of the number exposed in each social situation. Mortality records were analyzed by chi square.

Mortality of males from all causes was significantly higher in heterosexual pairs and groups ($p<.005$), which may be attributed to a higher level of interaction among males in the presence of females. Mortality of females from all causes increased stepwise with group size ($p<.005$), which must reflect the increasing intensity of social interaction as group size increased (*2*).

The total mortality for the experiment (both sexes and all ages through 45 weeks) was 24.6 percent. Leucosis, usually of the liver and spleen, accounted for 15.9 percent of the deaths. These values are in contrast with the results of earlier experiments in which the total mortality for 885 birds was 10.9 percent, and in which leucosis accounted for 4.8 percent of the deaths. The composition of the groups described here (two males to one female) was designed to increase the amount of social interaction. The frequency of leucosis under such conditions suggests that responses to the social environment also may influence, or determine, the degree of resistance to indigenous viruses and, in turn, the frequency of one form of malignancy in this race of chickens.

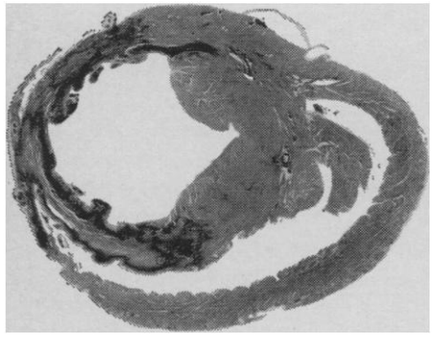

Fig. 1. Myocardial infarction in a male chicken aged 32 weeks, from a group of 24, consisting of 16 males and 8 females. Transverse section of the heart about midway between apex and base, dilated left ventricle to left, necrotic muscle (infarct), outlined by darkly stained zone, extends from midportion of anterior wall of left ventricle to midportion of intraventricular septum and to right ventricular wall. (Allochrome, × 2.5)

Infarcts of the myocardium were found in 21 males, all from cages that also contained females, and in 3 females from the cages that originally contained 24. The males died between weeks 16 and 45; and females between weeks 32 and 44.

The size of the infarcts ranged widely and were recognized macroscopically only in five instances. One of these involved a relatively large part of the left ventricular wall including part of the septum, and had led to dilatation of the chamber (Fig. 1). The remaining four were distinguished by focal discoloration of the muscle and by hemorrhage. Two of these extended through the entire thickness of the left ventricular wall.

Microscopic examination revealed equally extensive infarcts of the inner layers of the myocardium in two other instances. However, in more than half of this series the infarcts were about 2 to 3 mm in diameter, which means, of course, that all examples may not have been found even with multiple sections from two or more blocks that transected the heart.

All of the larger infarcts may be classed as recent, or at most, formed less than 12 hours before death. None of them were infiltrated by leucocytes. The smaller infarcts ranged more widely in apparent age. Many were recent, but in at least two hearts small cellular scars were associated with earlier stages of necrosis. The leucocytic response to myocardial necrosis was not pronounced in any instance.

This series of infarcts of the heart muscle was invariably associated with advanced grades of coronary arterial disease which, in no instance, was found to have caused complete obstruction. In fact, thrombi were found only in arteries and veins of older infarcts. Thus myocardial infarction in these chickens may be attributed to two concurrent factors: (i) rapidly developing coronary arterial disease which limited blood flow to the heart muscle, and (ii) demands for high levels of myocardial activity.

Leucosis has been suggested as a stimulus to, if not a primary cause of, coronary arterial disease in chickens (*3*). Our observations do not support this opinion. Instead, in this and earlier experiments leucosis has been associated with only minor grades of coronary disease. Further, the hearts of birds that died of leucosis were not regularly invaded by the malignant cells.

H. L. RATCLIFFE
R. L. SNYDER

Penrose Research Laboratory, Zoological Society of Philadelphia, and *Department of Pathology, University of Pennsylvania, Philadelphia*

References and Notes

1. H. L. Ratcliffe, T. G. Yerasimides, G. A. Elliott, *Circulation* **21**, 730 (1960); G. E. McKissick, G. L. Flickinger, H. L. Ratcliffe, *Federation Proc.* **20**, 91 (1961); H. L. Ratcliffe, in *Etiology of Myocardial Infarction*, T. N. James and J. W. Keyes, Eds. (Little, Brown, Boston, 1962), p. 61; H. L. Ratcliffe and R. L. Snyder, *Circulation* **26**, 1352 (1962).
2. G. L. Flickinger, *Gen. Comp. Endocrinol.* **1**, 332 (1961); G. L. Flickinger, thesis, Univ. of Pennsylvania.
3. J. C. Peterson and G. E. Cottral, *Arch. Pathol.* **49**, 699 (1950).
4. Supported by NIH grants HE 01979 and HE 04759.

19 March 1964