Nervous control of heart rate during excitement in the adult White Leghorn cock

JOHN L. TUMMONS AND PAUL D. STURKIE
Division of Physiology, Department of Animal Sciences, Rutgers University, New Brunswick, N. J. 08903

TUMMONS, JOHN L., AND PAUL D. STURKIE. Nervous control of heart rate during excitement in the adult White Leghorn cock. Am. J. Physiol. 216(6): 1437-1440. 1969.—The control of heart rate by the sympathetics and parasympathetics during excitement has been studied in the adult White Leghorn cock. The data presented in this study demonstrate that at the basal resting heart rate (BRHR) of 280-290 beats/min there appears to be present a significant amount of sympathetic and parasympathetic tone. This tone appears approximately balanced, as elimination of either nerve system resulted in approximately equal changes in the BRHR (increase or decrease). In the normal bird excitement-induced heart rate changes appear to result from a decrease in the resting vagal tone and increased sympathetic tone, with all immediate increases in heart rate above the BRHR level dependent on sympathetic tone. The normal BRHR can be maintained in the absence of extrinsic cardiac nerves. Complete denervation first becomes evident in the inability of the bird to significantly change its heart rate in immediate (0-6 sec) response to excitement.

Methods and Procedures

Determination of excitement response. The procedure used for the measurement of heart rate response to sudden excitement was carried out as follows: The animal was taken from its cage, weighed, and placed in an upright holder. Electrocardiograph electrodes were attached on both sides of the sternum and on each leg to record heart rate. The animal was then shielded from movement in the room by a paper flap installed on one side of the holder. Noise in the room was kept to a minimum throughout the determinations. The animal was allowed to remain in this condition for 15-20 min to provide ample time for the animal to adjust and reach a stable resting heart rate. At the termination of this period, heart rate was recorded and taken as the basal resting heart rate (BRHR). Normal and denervated animals were selected at random and then were excited suddenly by reaching over the paper flap, grasping the bird's comb, and shaking it for 2-3 seconds. Readings (beats/min) were counted for 6-set intervals taken immediately and after 10, 20, 30, 40, 60, and 120 sec postexcitement. Heart rate was recorded on a Grass model 5A recorder. All data were subjected to the Student $t$ test for the significance between two means.

Surgical sympathectomy. The procedure for the sympathectomies involved restraining the bird in a supine po-
operation, and anesthetizing the animal with sodium pentobarbital (30 mg/kg) through a right brachial vein canula. The animal was then placed on its keel in a V-shaped holder. The sympathetic nerve was exposed through an incision made between the scapula and intercostal muscle connecting the first to the second rib. From this view the sympathetic cardiac nerve is observed running parallel to a small vertebral vein (Fig. 1). The sympathetic cardiac nerve joins this vein shortly after its emergence from the first thoracic ganglion and courses with it to the point where the superior vena cava joins the heart. At this point the nerve turns toward the heart, joins the vagus nerve with its connective tissue investments and prevents damage to the ribs. Through the opening, provided for a minimum of pectoral muscle disturbance and precludes damage to the ribs. Through the opening, the vagus nerve with its connective tissue investments were exposed lying along the superior vena cava (Fig. 1). The nerve was freed at the level of the ganglion nodosum to above the pulmonary artery. At this time all fibers except the recurrent vagi going over the pulmonary artery, and including the superior cardiac vagal nerve, were tied and sectioned. The central ends were then placed in a polyethylene tube which extended above the intercostal wall. The nerve was then tied to the pectoral muscle. The spreaders were removed and the incision closed. The same operation was subsequently performed on the opposite side and the bird was treated with an antibiotic as previously described.

Atropine administration. After determining the basal resting heart rate of the control bird, atropine sulfate (0.25 mg/kg) was injected via the brachial vein catheter and the heart rate was determined 2–5 min later.

RESULTS

Fifty-two adult single-comb White Leghorn cocks were used in this experiment. They consisted of: controls, 18 birds; sympathectomized, 10 birds; vagotomized, 8 birds; atropinized, 6 birds; and completely denervated, 10 birds. Basal resting heart rate, along with heart rate changes produced by excitement, were studied approximately 6–7 days after the surgical denervations. The reason for this time delay was threefold. The first was to establish a standard time for comparison of different groups, the second was to allow for recovery so as to minimize surgical effects, and the third was to allow enough time lapse after denervation for the depletion of postganglionic nerve fibers. Since this was the first attempt at surgical denervation in birds, the time interval was obtained from observations on the general health of the animals after surgery and from comparable studies in mammals. Total depletion of norepinephrine (NE) stores in the heart occurs within 3–5 days after total sympathectomy in the dog (6, 17), cat (20), and baboon (23). This depletion of NE stores appears to be consistent with degeneration of the severed postganglionic fibers. Since the bird has a higher body temperature, and thus higher metabolism than the dog, cat and baboon, it was presumed that 6–7 days after sympathectomy would be a satisfactory time for measurement.

This was borne out by histochemical determination of catecholamines in the bird heart after this interval. The results (unpublished observations) indicate that the sympathetic nerve terminals in the bird heart are essentially degenerated 7 days postsympathectomy.

Sympathectomy. Sympathectomy alone produced a 19–20% decline in heart rate from the BRHR of the control animals. Variability of the BRHR of the sympathectomized birds was reduced to approximately 64% of that observed in the control animals (Table 1). Postexcite-
ment heart rate (280 beats/min) was not significantly different from the BRHR of the control and completely denervated birds (Table 1). At basal heart rates up to approximately 286 beats/min normal birds after excitement demonstrated an average increase in heart rate of 100 beats/min above that shown by sympathectomized birds (Fig. 2). The sympathectomized animals appeared totally unable to raise their heart rates in excess of 286 beats/min. However, normal birds at a basal heart rate of 286 beats/min were still capable of elevating their heart rate 100 beats/min. It is observed from Figs. 2 and 3 that the increase in heart rate of a sympathectomized bird is limited to the level of the control BRHR and is thus unable to exceed it. Therefore, the total postexcitement increase in heart rate above the BRHR of the normally innervated bird is completely dependent on sympathetic tone. The 20% decline in resting heart rate following sympathectomy is attributable to the absence of sympathetic tone and the equal increase in heart rate following excitement after sympathectomy results from decreased vagal tone (Fig. 3).

**Vagotomy.** Vagotomy produced a significant increase (90–91%) in the normal BRHR (Fig. 3). A similar change was produced in atropinized birds (Table 1). Stimulation of the cervical vagus above the original transection had no effect on heart rate in two representative birds, thus demonstrating the effectiveness of the original surgical procedure and the lack of nerve regeneration. The surgically vagotomized birds lived for 2 months, after which they were sacrificed. During this time there was no incidence of sickness and the health of these animals appeared not to be different from normal healthy birds. They were able to attain the same maximum heart rate level (387 beats/min) after excitement as the control excited birds (383 beats/min), Fig. 3. The percent increase of the two groups, however, was different. The heart rate of excited vagotomized animals increased 13–14% above their resting rate; this is attributable solely to increased sympathetic tone.

**Complete denervation.** A summary of the heart rate data obtained in this experiment along with the significance between heart rate levels can be found in Table 1. From this table it can be observed that the basal resting heart rate level (BRHR) of the normal control birds is 284 beats/min. This heart rate remains unchanged by complete denervation (284 beats/min). A comparison of the coefficients of variation between these two groups demonstrates that complete denervation reduces the variability in the basal heart rate to approximately 33% of that found in the control bird. The control group increased its heart rate to 383 beats/min, but the completely denervated birds were unable to significantly increase their heart rate (290 beats/min). It is of interest to note that in the control group, postexcitement variation in heart rate was reduced to approximately 34% of the variability found at the normal BRHR level, whereas the completely denervated group demonstrated the same level of variation before and after excitement (Table 1).

**DISCUSSION**

These data suggest that the BRHR of the normal bird can be maintained in the absence of extrinsic cardiac
nerves. The most apparent manifestation of complete denervation is the inability of the animal to instan-
taneously change its heart rate in response to excitement;
the normal bird increases its heart rate approximately
34–35% above its normal BRHR when excited.

The approximately equal changes in BRHR (although opposite in direction) produced by sympathectomy and
vagotomy, as well as the return to the normal BRHR
following both operations, demonstrate that in the intact
animal there is a balance in amount of sympathetic
excitement are mainly dependent upon sympathetic tone.

In the sympathetically denervated birds elevates the heart rate only to the
level of the BRHR of the control and completely de-
nervated ones and never above it, and this increase in
heart rate is attributable to decreased vagal tone. All
increases in heart rate above the normal BRHR following
excitement are mainly dependent upon sympathetic tone.

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norepinephrine in heart rates (15). In some dogs vagal restraint is believed
to predominate in the control of heart rate (13, 21). Re-
cent evidence, however, by Adolph (1) in rats suggests
a relatively equal resting tone of both the vagus and ac-
celerators which agrees with our data on chickens.

The fact that the resting heart rate of the completely
denervated chicken (280–290 beats/min) approaches
that of the intact innervated one indicates that intrinsic
heart rate is determined or regulated by nonneurogenic
factors such as body temperature, hormones, and met-
abolic state (2).

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