THE ROLE OF FEATHER MUSCLE RECEPTORS IN INTRAFOLLICULAR PRESSURE AND FEATHER RELEASE

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Ronald Arthur Peterson

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Robert K. Ringer
Major professor

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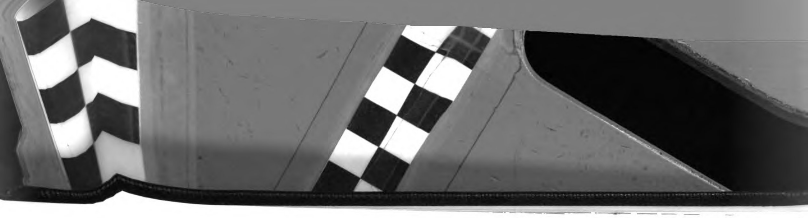

ABSTRACT

THE ROLE OF FEATHER MUSCLE RECEPTORS IN
INTRAFOLLICULAR PRESSURE AND
FEATHER RELEASE

by Ronald Arthur Peterson

Previous investigations have indicated that nerve fibers of the autonomic nervous system innervate the feather muscles. This research was undertaken to study the effects of the feather muscles on feather release and feather shaft movement when stimulated by various neuromimetic drugs.

By direct cannulation, the intrafollicular pressure within an individual feather follicle from the caudal or femoral feather tracts of S. C. White Leghorn hens was recorded. The force necessary to pull a feather from its follicle (feather pulling force) was measured simultaneously.

When the birds were anesthetized, intrafollicular pressure and feather pulling force decreased. After death, however, the feather pulling force and intrafollicular pressure did not appear to be related. The feather pulling force increased until the feathers were considered in a tightened state (1 to 3.3 minutes after death) and remained constant thereafter. Intrafollicular pressure fluctuated from the time of death of the bird until the end of the experiment 112 minutes later.

After comparing the effects of various neuromimetic drugs used in the anesthetized bird, it appears that while both cholinergic receptor stimulating drugs (pilocarpine,

carbachol, physostigmine and acetylcholine) and adrenergic alpha receptor stimulating drugs (norepinephrine, epinephrine and ephedrine) caused an increase in intrafollicular pressure, the cholinergic class of drugs were more pronounced in causing the tightened state to occur within the feather follicle than were the adrenergic drugs.

Adrenergic beta receptors were also studied using isoproterenol (beta stimulating) and phenoxybenzamine (alpha inhibiting, to reduce chances of alpha stimulation). The data indicated that beta receptors were not an important factor in regulating intrafollicular pressure, feather release and feather shaft movement.

Adrenergic alpha stimulating drugs caused the feathers to be depressed close to the body, while cholinergic drugs caused the feathers to become erected in the femoral feather tract. Thus, in the chicken, the feather muscles appear to have both adrenergic alpha and cholinergic receptors.

THE ROLE OF FEATHER MUSCLE RECEPTORS IN
INTRAFOLLICULAR PRESSURE AND
FEATHER RELEASE

By

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INTRODUCTION

One of the major problems facing the poultry processing industry today is that of obtaining a maximum, top quality meat product for marketing. With present-day methods of processing there is a substantial loss in the quality of the product, in that scalding tends to partially cook the skin, thus reducing shelf life. In many cases, when the feathers are removed from birds by mechanical pickers, the skin is torn and bones are broken which in turn lowers the quality and value of the product.

Although there has been much research in the area of feather removal, little is known about the particular forces or mechanisms which hold the feather in its follicle. It would be logical to have a thorough understanding of these mechanisms before one undertakes to improve present-day methods of feather removal.

The purpose of the investigations presented in this thesis was to obtain basic information or insight into the physiological mechanism(s) which is/are responsible for holding the feather in its follicle both before and after death.

REVIEW OF LITERATURE

The Anatomy of the Feather Follicle and the Muscles which Move the Feather

When reviewing literature, one finds that there is very little information on the anatomy of the feather follicle and the muscles which move the feather.

Feather muscles apparently were first discovered by Nitzsch and Burmeister (1840). Only a brief description was given. Sauffert (1862) described the muscles in the skin of the bird as being unstriated and with each feather follicle having two or four separate muscles. The muscles were described as being connected to the follicle with elastic tendons. These muscles were observed to course from the upper part of one follicle to the lower part of a neighboring follicle.

Helm (1886) also found that there were generally four smooth muscles attached to a single feather follicle, although in some cases, the number may be as high as six. He also noted that the size of the muscles varied directly with the amount of movement and that these muscles were involved in either feather ruffling or depression.

Langley (1902b) observed that each feather follicle had two sets of muscles which he designated as erectors and depressors. He noted that the number of muscles varied to as high as 16 attachments per follicle. The striated cutaneous muscles of the neck region were also discounted as the sole effector in the erection of feathers. In a subsequent and more thorough investigation, Langley (1904) observed that the feathers were supplied with a complicated system of smooth muscles, which he divided into three classifications: (1) erector muscles passing from the neck of a follicle to the base of a follicle in an anterior direction from the first, (2) depressor muscles passing from the neck of one follicle to the base of a follicle in a posterior direction from the first, (3) retractor muscles which pass from the neck of one follicle to the neck of another.

Dreyfuss (1937) reported that the feather follicle has both erector and depressor smooth muscles. He also described the nervous and vascular supply to the feather muscles and follicles. The blood vessels and nerves form sort of a complex tent which surrounds the lateral and superficial surfaces of the muscles, with innervation only occurring at their dermic insertions. Only the collateral branches of the nerve trunk are motor to the muscles. Sources of nerves in the follicle wall were described as such: (1) papilla--receives nerves from the principal vasculo-nervous axis; (2) middle--is supplied with branches from the afferent nerves of the feather muscles; (3) top--is rich in sensory nerves of the skin. The innervation becomes more dense toward the collar. The permanent papilla was described as having ganglion cells which migrate into the growing feather shaft along with sympathetic fibers during feather growth.

Lillie (1940), when describing growing feather follicles, depicted the layer which forms the follicular wall immediately adjacent to the keratinized epithelium as being muscular. To the contrary, Ostmann, et al. (1963a) examined histological sections of the feather follicle stained with specific differentiating stains and concluded that the follicular wall is not muscular, but is composed mainly of connective tissue. This connective tissue was found to have a high content of elastic fibers. These workers also indicated that the feather follicles are supplied with a complex system of smooth muscles which are attached to the follicular wall by elastic tendons. The follicle and its immediate area were found to be supplied with a rich supply of nerves. Some of the nerve endings within the smooth muscle were demonstrated to be of a cholinergic nature.

Stettenheim, et al. (1963) indicated that nonstriated feather muscles in the dermis were principally responsible for adjusting the posture of the feather. Generally, there are four pairs of muscle connected to each feather follicle, although the number of muscles per follicle may vary dependent upon location and feather tract. They observed that each follicle is usually connected by muscle bands to four adjacent follicles and in each set of muscles there is an erector and larger depressor muscle which cross each other.

The Nervous System and Its Relationship to Feather Movement and Release

Although Sauffert (1862) indicated that the feather muscles were under involuntary control, Langley (1902a, 1902b, 1904) was the first to make a thorough study of the innervation of the feather.

Langley (1902a) first noted, after the cervical spinal cord was severed and the lower end stimulated, that the contour feathers over the entire body were drawn close to the body surface. The nerves which were stimulated leave the spinal cord, enter the ganglia of the lateral chain and synapse with post ganglionic fibers which then course to the skin. He observed that, after the cervical sympathetic was sectioned, the feathers under the influence of this nerve became ruffled and somewhat erected. When nicotine was injected intravenously, the contour feathers over the entire body became depressed. When the cervical sympathetic was stimulated in nicotine pre-treated birds, there were some cases in which the feathers became erected instead of depressed. Upon further examination, Langley (1902b) discounted the striated cutaneous muscles as a sole effector in the erection of feathers. He noted, after spinal cord section and stimulation and also immediately post-mortem, that rhythmic erection and depression

of groups of feathers occurred randomly over the entire body. Later, Langley (1904) indicated that the larger depressor and smaller erector muscles were both supplied by sympathetic nerves. When the cervical sympathetic was stimulated, the feathers were generally depressed, but occasionally erection occurred. Section of either preganglionic or post ganglionic sympathetic fibers resulted in erection of the feathers. After treatment with curare no effects were observed; however, in the same birds, strychnine was later given and this combination treatment resulted in irregular rhythmic depression and erection of the feathers upon spinal cord stimulation. Langley also found that nicotine stimulates the sympathetic ganglia, while on the other hand, the preganglionic fibers are not paralyzed. Atropine, apocodeine and adrenalin were observed to have little or no effect on movement of the feather muscles.

Probably the first investigation of feather release was by King (1920). By brain sectioning and electrical stimulation of various areas of the exposed brain, he concluded that the brain center involved in the control of feather release was located in the medulla. Also, this author, when using the following drugs, chloroform, atropine, scopolamine, apomorphine, strychnine and amyl nitrite, found that the feathers were loosened. Drugs such as morphine, eserine, adrenalin, curare, heroin, chloral hydrate, emetine or camphor, have no effect on feather loosening.

King also found that electrical stimulation of the medulla caused feather loosening.

To the contrary, Weaver (1936) found, when using birds killed by brain piercing through the eyesocket and also by brain dissection in anesthetized birds, that the feather release center was located at the anterior base of the cerebellum.

Later, Rose (1939) developed a successful method for feather release which incorporated an electrical shocking device. Electrodes were placed on the outer surface of the head below the earlobes in such a manner as to allow the electrical current to flow through the forward base of the cerebellum.

When studying the effect of reserpine (Serpasil) on adult White Leghorn capons, Sturkie, et al. (1958) noted that after treatment there was an increase in shedding of feathers during handling. These authors suggested that a relaxing effect occurred in the feather follicle.

Sodium pentobarbital, a general anesthetic, was used by Huston and May (1961) to loosen the feathers of broilers. They found, after the birds were bled and dry picked in a mechanical picker, that 90 to 95 percent of the feathers were removed.

Klose, et al. (1961) found, after birds were treated with sodium pentobarbital and reserpine, that there was a marked reduction in feather pulling force. Reserpine had no appreciable carry-over effect after death. In addition, Klose, et al. (1962) reported that brain sticking reduced feather pulling force, but this effect lasted for less than one minute. These authors found that when birds were given a minimal dose of sodium pentobarbital feather pulling force rose rapidly to pre-treatment values after death, while massive lethal doses prolonged the time post-mortem that the feather pulling force remained below the pre-tested level.

Feathers were loosened (Ostmann, et al., 1963b) by both local and general anesthetics, by tranquilizing drugs such as chlorpromazine and promazine and by the neuromimetic blocking drugs atropine and yohimbine. On the other hand, neuromimetic drugs had no effect in reducing feather pulling force. In addition, Ostmann, et al. (1964) observed that all levels of spinal transection significantly reduced feather pulling force posterior to the level of transection. Electrical stimulation of the distal part of the severed spinal cord resulted in a marked increase in feather pulling force posterior to the level of transection.

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OBJECTIVES

The present investigation was undertaken:

1. To develop a technique for measuring pressure (intrafollicular pressure) within a feather follicle.
2. To measure and compare intrafollicular pressure of feather follicles in both the loose and tight states.
3. To compare the effects of several adrenegic and adrenergic blocking agents on intrafollicular pressure, feather release and feather shaft movement and to use the above data to determine if the feather muscles have adrenergic alpha and/or beta receptors.
4. To compare the effects of several cholinergic and cholinergic blocking agents on intrafollicular pressure, feather release and feather shaft movement and to use the above data to determine if the feather muscles have cholinergic receptors.

EXPERIMENTAL PROCEDURE

In developing a technique for the measurement of intrafollicular pressure (IFP), mature S. C. White Leghorn-type hens, reared at the Michigan State University poultry farm, were used. The birds were placed in a prone position on an operating cradle with the feet secured in a posterior position. This technique held the birds in such a manner as to virtually eliminate struggling. The brachial vein in the right wing was then cannulated (procaine was used as a local anesthetic by injecting subcutaneously) with an I.D. 0.030 inch x O.D. 0.048 inch polyethylene cannula attached to a 6 ml plastic syringe. The above system was subsequently used to anesthetize the birds with either sodium pentobarbital or sodium phenobarbital.

A mature feather was randomly selected from either the femoral or caudal feather tract for studying IFP. All of the feathers in the area of the selected feather shaft were cut at the level of the epidermis leaving the selected feather shaft unobstructed. A piece of fine thread was then stitched superficially into the epithelium in a purse string manner around the selected mature feather shaft (Figure 1 and 2). The feather shaft was then pulled and the empty follicle filled, using a syringe with a blunt 22 gauge needle, with S.A.E. No. 20 oil or mineral oil. The reason for using oil

Figure 1. A photograph of a S. C. White Leghorn hen showing, by the use of India ink, the outline of the femoral feather tract and the individual feather follicles. The femoral feather tract was used for the measurement of intrafollicular pressure under various physiological conditions.

Figure 2. This photograph demonstrates how a fine thread was stitched into the epithelium in a purse string manner around the feather shaft.

in the feather follicle was an attempt to eliminate any diffusion or seepage of liquid from the follicle through the epithelium of the feather follicle. In previous attempts, when using only physiological saline, loss of fluid through the skin resulted in failure to record changes in IFP. A polyethylene cannula (described below), filled with water, was then inserted into the feather follicle. The purse string was then drawn tight and tied (Figure 3). The cannula was in turn attached to a Statham model P23BB venous transducer, thus forming a closed hydraulic system. An I.D. 0.023 inch x O.D. 0.038 inch cannula, with a 22 gauge metal tip inserted into its free end to prevent occlusion whey tying it into the follicle, was employed in measuring IFP in the femoral tract, while an I.D. 0.047 inch x O.D. 0.067 inch cannula without a metal tip was used in the retrice feather follicles of the caudal tract. All recordings were made on a Grass Model 5 polygraph.

After the purse string was tied tightly around the cannula, approximately 15 to 20 mm Hg of pressure were applied to the system. The IFP would drop somewhat following the application of the initial pressure into the system. In each case, the initial pressure decreased slightly and then leveled out over a one minute period to a value that was considered as the base pressure in the pre-anesthetized bird. All subsequent pressures were recorded as a percent of this base. In some cases, the head of the bird was

Figure 3. After removal of the feather shaft, oil was injected into the feather follicle and then the cannula was inserted with the purse string being drawn tight and tied as seen in this photograph.

covered with a paper towel to avoid frightening it since movement would induce a marked fluctuation in the pressure of the system.

In preliminary trials, the number four retrice feather of the caudal feather tract, counting from the end of the row, was arbitrarily selected for the measurement of IFP. In subsequent trials, follicles used for pressure recordings were arbitrarily selected from the distal end of the femoral feather tract.

Throughout the various phases of the experiment, feathers were pulled from the dorsal feather tract to determine whether or not they were in the tightened state. The force necessary to pull a feather from its follicle (feather pulling force or FPF) was measured using a spring scale (Klose, et al., 1961) throughout the trials. Feather pulling force was compared to IFP changes. Feathers requiring more than 500 grams of force to be pulled from their follicle were considered "tight"; those requiring less than 130 grams were considered "loose."

Birds were slowly infused through the brachial vein with either 3 percent sodium pentobarbital or 10 percent sodium phenobarbital until the desired plane of anesthesia was reached. A light plane of anesthesia, as defined by Fedde, et al. (1963), was used. At this plane of anesthesia a chicken responds to pinching of the comb, but shows no response to the pinching of the skin or toes. Due to marked individual variations in response to anesthesia, it was

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necessary to give the anesthetic "to effect." The amounts given ranged from 115 to 140 mg/kg for sodium phenobarbital and 25 to 45 mg/kg for sodium pentobarbital. Since sodium phenobarbital appears to have a more lasting effect, sodium pentobarbital was used only in Experiments 1, 2, 3, 21, and 22.

Simultaneously with IFP, blood pressure was measured (exception: preliminary phases) with a Statham Model P23 AC arterial transducer, by direct cannulation, using an I.D. 0.034 inch x O.D. 0.050 inch cannula connected to the same Grass polygraph as for IFP, of the ischiatic artery in the area of the thigh. The ischiatic artery was exposed by an incision along the distal edge of the femoral feather tract (opposite to the tract where IFP was measured) and by separating the *M. biceps Femoris* from the *M. semimembraneous* and *M. semitendinosus*. Blood pressure was used only as a criteria to evaluate the effectiveness of the drugs employed. Since blood vessels contain smooth muscle and also due to the fact that the feather muscles are of the smooth type, it was assumed that when the drugs influenced the blood vessels, that the feather follicles would also be affected.

All drugs were injected intravenously as rapidly as possible through the opposite uncannulated brachial vein "to effect," that is, until a response was observed either in blood pressure and/or IFP. Drugs and average dosages used were as follows:

a. Cholinergic--pilocarpine nitrate 5.6 mg/kg, carbachol 0.152 mg/kg, acetylcholine chloride 0.466 mg/kg, physostigmine salicylate 0.132 mg/kg.

b. Cholinergic blocking--atropine sulfate 2.83 mg/kg.

c. Adrenergic--epinephrine 0.043 mg/kg, norepinephrine 0.112 mg/kg, 0.295 mg/kg, and 0.375 mg/kg, ephedrine 5.3 mg/kg.

d. Adrenergic beta--isoproterenol hydrochloride 1.02 mg/kg.

e. Adrenergic alpha blocking--phenoxybenzamine 39.1 mg/kg.

f. Adrenergic beta blocking--dichloroisoproterenol 6.5 mg/kg.

g. Ganglionic blocking--hexamethonium chloride 7.3 mg/kg.

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RESULTS

Caudal Feather Tract

Experiment 1.--The effects of anesthesia and death by cervical dislocation on intrafollicular pressure and feather release.

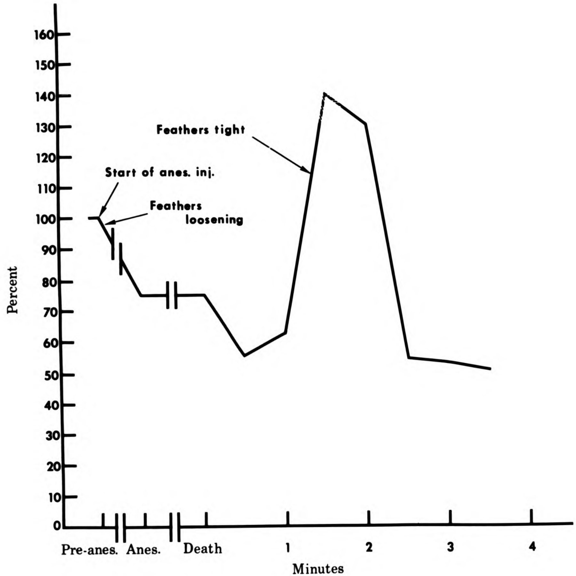
In Experiment 1, intrafollicular pressure (IFP) was measured (for details see Experimental Procedure) in the number four retrice feather follicle of the caudal feather tract of 11 birds. To avoid movement, the fleshy base of the external tail was secured to a ring stand with a towel forceps, which would have caused pressure changes within the recording system. Following anesthetization by 3 percent sodium pentobarbital, the IFP decreased to 76 percent of the preanesthetized base value of 100 percent (Figure 4). The force necessary to pull the feather from its follicle, known as feather pulling force (FPF), was measured either in the femoral or dorsal feather tracts, and decreased until the feathers were considered loose (FPF of less than 130 grams). The FPF decreased more rapidly than the IFP following anesthetization. After the birds had been anesthetized for 1 to 2 minutes, they were killed by cervical dislocation. This was accomplished by using a bone cutting forceps to crush several of the cervical vertebra. Thirty seconds (30) after cervical dislocation IFP decreased to 56 percent, while the FPF indicated that the feathers were in a loosened

Figure 4. The effects of anesthetization and death (11 mature S. C. White Leghorn hens were used) by cervical dislocation on intrafollicular pressure in the number four retrice tail feather (counting from the end) of the caudal feather tract.

1. Data were based on a 100% control pressure before the birds were anesthetized.

2. The arrow indicates when the feather pull resistance was increasing.

3. The area depicted by ┬ ┬ ┬ ┬ ┬ ┬ ┬ indicates intrafollicular feather pressure higher than the recording capacity of the machine (as it was set).



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state. About one minute after cervical dislocation, the birds went into mass spasmodic muscle contractions which lasted from 1 to 2 minutes. During this time, the feather shafts raised and lowered irregularly over the entire body. When the spasmodic contractions began, the FPF increased as did the IFP, which rose to 139 percent. Approximately 86 seconds after death, The FPF had increased to over 500 grams and the feathers were considered tight. The feathers remained in this tightened state, regardless of the IFP level, during the rest of the experiment. When the spasms stopped, IFP had decreased to 54 percent.

Femoral Feather Tract

Experiment 2.--The effects of anesthesia and death by cervical dislocation on intrafollicular pressure and feather release.

IFP was measured from feather follicles arbitrarily selected in the femoral feather tract of 15 birds. FPF was measured from feathers arbitrarily selected in the opposite femoral feather tract and compared with IFP throughout the experiment. Following anesthetization, IFP decreased to 65 percent from the 100 percent pre-anesthetized base pressure and the FPF decreased until the feathers were loose (Figure 5). As in the previous experiment, the FPF decreased more rapidly than did the IFP following anesthetization. After 1 to 2 minutes in the anesthetized state the birds were killed by cervical dislocation. One minute after death, the IFP had increased to 100 percent, while the feathers remained

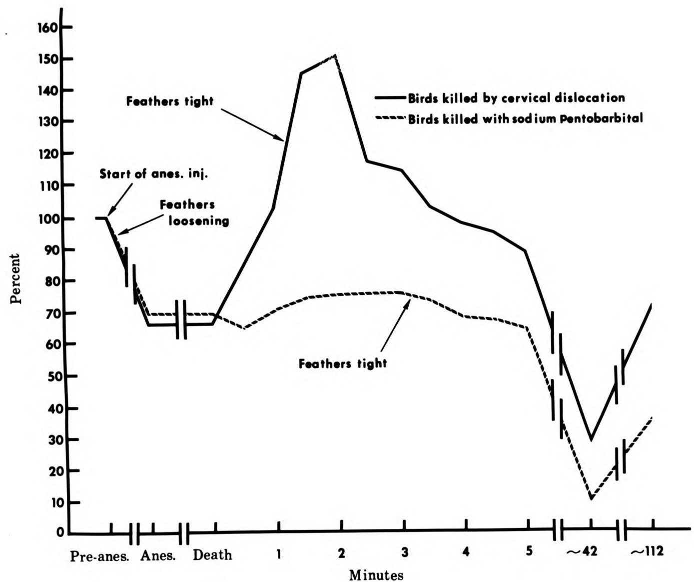
Figure 5. The effects of anesthetization and death by cervical dislocation (15 mature S. C. White Leghorn hens) and death by an overdose of anesthesia (5 mature S. C. White Leghorn hens) on intrafollicular feather follicle pressure in feathers selected arbitrarily in the femoral feather tract.

1. Data were based on a 100% control pressure before the birds were anesthetized.

2. Solid ———— indicates birds dispatched by cervical dislocation, ┬ ┬ ┬ ┬ ┬ ┬ indicates where intrafollicular pressure increased beyond the recording capacity (as it was set) of the machine.

3. Dashes ------ indicates birds which were dispatched by an overdose of 3% sodium pentobarbital.

4. The arrows indicate when the feather pull resistance was increasing.


Birds killed by cervical dislocation
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Feathers tight
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loose. Approximately one minute after cervical dislocation, the birds went into mass spasmodic muscle contractions similar to those previously described. IFP increased to 150 percent of the base pressure during this time period. During the spasms, which continued for 88 seconds after cervical dislocation, the force necessary to pull the feathers increased until the feathers were tight (500 grams or over). The feathers remained tight during the remainder of the experiment irrespective of the IFP level. IFP decreased to 97 percent after the spasms. Small irregular pressure changes continued for 52 minutes after cervical dislocation. Although the IFP fell to 28 percent in an average of 39 minutes after death, the feathers remained tight. Approximately 120 minutes after death, IFP increased to 69 percent.

Experiment 3.--The effects of anesthesia and death by sodium pentobarbital on intrafollicular pressure and feather release.

In this experiment, the same procedure was used as in Experiment 2 except that the 5 birds were killed with an overdose of 3 percent sodium pentobarbital. Death was considered to occur at the time of respiratory failure. When the birds were anesthetized to a level at which there was no response when the comb was pinched, IFP decreased to 69 percent of the 100 percent pre-anesthetized base pressure (Figure 5). FPF also decreased to a level at which the feathers were considered to be in the loosened state. After the bi

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the birds were anesthetized for 1 to 2 minutes, they were killed with an overdose of anesthetic. No mass spasmodic muscle contractions were observed, while IFP increased to 74 percent. The FPF did not increase until 3 minutes after death. The feathers remained tight from this point on regardless of the IFP. Concurrent with the increase in FPF were small sporadic pressure changes (similar to those observed in Experiment 2) which continued for approximately 60 minutes after death. IFP decreased to its lowest level (10 percent) 44 minutes after death with the feathers still remaining in a tightened state. IFP then increased to 34 percent of the pre-anesthetized base pressure in an average of 103 minutes after death.

Immediately after the birds (Experiments 1, 2, and 3) were given a general anesthetic (sodium pentobarbital), IFP decreased and simultaneously the feathers entered the loosened state. Following death, the IFP and FPF were apparently not related, since after the FPF had increased to where the feathers were considered tight, they remained tight regardless of the IFP level.

The Effects of Cholinergic Drugs and Cholinergic Blocking Drugs on Intrafollicular Pressure and Feather Release

The cholinergic drugs and blocking drugs listed in the experiments below, were used to determine whether or not the receptors of nervous innervation on feather muscles are of a cholinergic nature. The effect of these drugs on IFP and feather release was studied.

Experiment 4.--Pilocarpine nitrate (6 birds).

Pilocarpine nitrate, a synthetic drug, produces strong cholinergic post ganglionic stimulation. This drug also produces a slight amount of ganglionic stimulation. Pilocarpine's main mechanism of action is by direct stimulation of cholinergic receptors in smooth muscle (Cutting, 1964). Pilocarpine was used in the following experiment to determine if the feather muscles have cholinergic receptors and how stimulation of these receptors might affect the feather release mechanism.

After the initial 100 percent base IFP was established, the birds were anesthetized with 10 percent sodium phenobarbital, resulting in a decrease of IFP to a 60 percent level (Figure 6). FPF also decreased to a level at which the feathers were considered to be in the loosened state after anesthetization. Ten (10) seconds after the start of injection of pilocarpine (5.60 mg/kg), IFP increased to a 120 percent level. The IFP indicated that the feathers entered the tightened state within 23 seconds after the start of injection and remained as such for the duration of the experiment. IFP continued to increase until it reached a peak of 146 percent which occurred in 60 seconds from the start of injection. At the termination of the experiment, 120 seconds after the initial injection, IFP was 141 percent of the original base pressure.

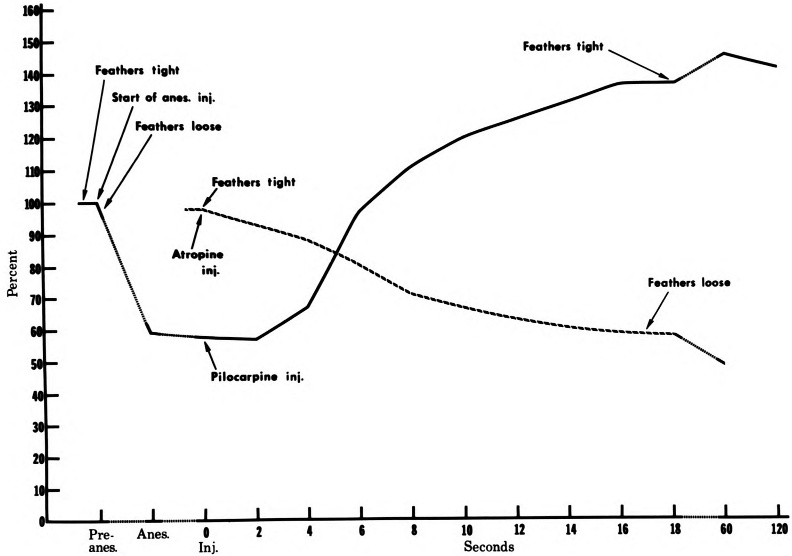
When pilocarpine was injected, IFP increased the feathers originally in the loosened state entered the

Figure 6. The effects of cholinergic and cholinergic blocking drugs on intrafollicular pressure within feather follicles arbitrarily selected in the femoral feather tract. All percentages given are in relationship to the intrafollicular pressure prior to anesthetization or drug treatment (100% as a base).

Drug used:

Pilocarpine nitrate (cholinergic) (6 birds used) 5.6 mg/kg ————————

Atropine sulfate (cholinergic blocking) (6 birds used) 4.3 mg/kg ------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tightened state. These data indicate that the feather muscles have cholinergic receptors and that they influence IFP and cause feather tightening.

Experiment 5.--Atropine sulfate (6 birds).

The birds in this experiment were given atropine to determine if the cholinergic receptors of feather muscles could be blocked and what effects this might have on feather release.

Atropine sulfate, a synthetic anticholinergic agent, produces cholinergic inhibition and depresses basal ganglia. The antropine molecules apparently compete with the cholinergic stimulating drugs to attach themselves to only cholinergic post ganglionic receptors, thus preventing stimulation of the smooth muscle by blocking the access of acetylcholine to the muscle receptors (Cutting, 1964).

The IFP level in the untreated bird was considered as the 100 percent base pressure level (Figure 6). After the injection of atropine, IFP decreased rapidly during the first 12 seconds to a 63 percent level. The feathers entered the loosened state in 17 seconds and remained in this state for the duration of the experiment. Sixty (60) seconds after the initial injection of atropine (2.83 mg/kg), IFP decreased to 48 percent of the original base pressure.

The injection of atropine into the unanesthetized bird resulted in a decrease in IFP and loosening of the feathers. This drug apparently blocked the cholinergic receptors of

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the feather muscles, thus causing the feathers to enter the loosened state.

Experiment 6.--Pilocarpine nitrate followed by atropine sulfate (6 birds).

The combined effects, of the two drugs used in the two previous experiments, on the cholinergic receptors, were examined in the following experiment.

Since pilocarpine stimulates the cholinergic receptor sites on smooth muscle and atropine acts as a direct antagonist, these two drugs were used in tandum in this experiment and their effects upon IFP and feather release were observed. In this experiment, blood pressure was also measured simultaneously with IFP and FPF. Blood pressure was used as an indicator of the effectiveness of the drugs administered.

After anesthetization with 10 percent sodium phenobarbital, the IFP decreased to 33 percent of the original base pressure (example, Figure 7-A). Simultaneously, the feathers entered the loosened state and a decline was also noted in blood pressure.

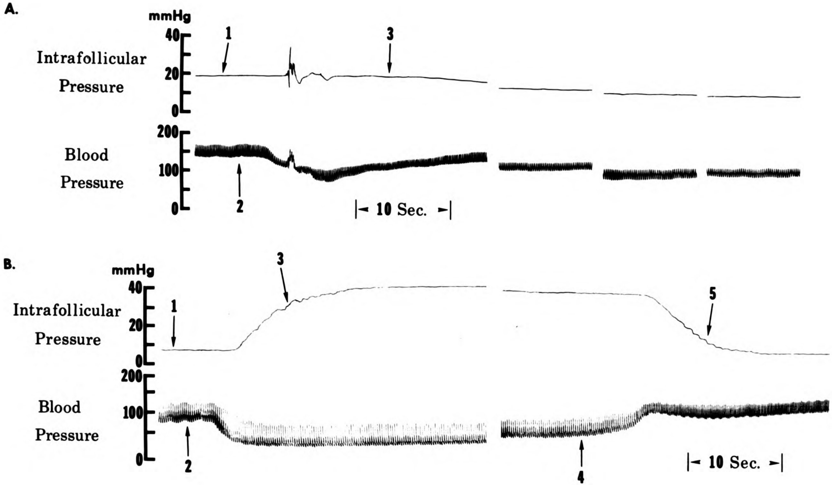
Two (2) seconds after the injection of pilocarpine (5.60 mg/kg), IFP increased and reached 191 percent in 22 seconds (example, Figure 7-B). The feathers entered the tightened state in 13 seconds after injection. Blood pressure decreased almost immediately after the injection of pilocarpine. Approximately 80 seconds after the injection of pilocarpine, atropine in the form of a sulfate

Figure 7-A. The effect of sodium phenobarbital (an anesthetic), when injected "to effect," in intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from a S. C. White Leghorn hen. Blood pressure was recorded as an indication of effect. Each time break is equal to 30 seconds.

1. Feathers in tightened state.
2. Anesthetic injected.
3. Feathers in loosened state.

Figure 7-B. The effects of pilocarpine nitrate and atropine sulfate, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indication of effect. The time break is equal to 30 seconds.

1. Feathers in loosened state.
2. Pilocarpine (cholinergic, 5.60 mg/kg) injected.
3. Feathers in tightened state.
4. Atropine (cholinergic blocking, 2.83 mg/kg) injected.
5. Feathers in loosened state.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(2.83 mg/kg) was injected. IFP decreased from 176 percent to 42 percent in 22 seconds following atropine injection. The feathers entered the loosened state in 13 seconds after the injection, while blood pressure increased.

When pilocarpine was injected, the cholinergic receptors of the feather muscles were apparently stimulated and IFP increased, while the feathers entered the tightened state. Atropine directly antagonized the effects of pilocarpine by blocking the cholinergic receptors resulting in a decrease in IFP and loosening of the feathers. Thus, these data indicate that cholinergic receptors are involved in the feather release.

Experiment 7.--Carbachol followed by atropine sulfate (6 birds).

To obtain additional evidence that cholinergic receptors are involved in feather release, carbachol was used.

Carbachol is also a synthetic cholinergic stimulating drug which produces ganglionic, muscular and post ganglionic effects (Cutting, 1964). Atropine was used to antagonize the effects of carbachol on IFP and feather release. Blood pressure was again used to indicate the effectiveness of the drugs used.

Following the injection of the anesthetic, 10 percent sodium phenobarbital, IFP decreased to 50 percent of the original base pressure (example, Figure 7-A). Blood pressure also decreased as did the FPF.

After the injection of carbachol (0.152 mg/kg), IFP increased to 163 percent within 22 seconds (example, Figure 8-C). The feathers entered the tightened state 16 seconds after the injection of carbachol, while blood pressure decreased within 10 seconds. Approximately 61 seconds after the injection of carbachol, atropine (2.83 mg/kg) was injected. IFP then decreased from 133 to 45 percent within 14 seconds and concomitantly the feathers entered the loosened state. Blood pressure increased within 6 seconds after atropine was injected, but did not remain at this level, as there was a gradual decline during the remainder of the experiment.

The effects of carbachol, which stimulates the cholinergic receptors of smooth muscle, were to cause an increase in IFP and to result in the feathers entering the tightened state. These effects were reversed by atropine, thus indicating the presence of cholinergic receptors on feather muscles.

Experiment 8.--Physostigmine salicylate pre-treatment followed by acetylcholine chloride (6 birds).

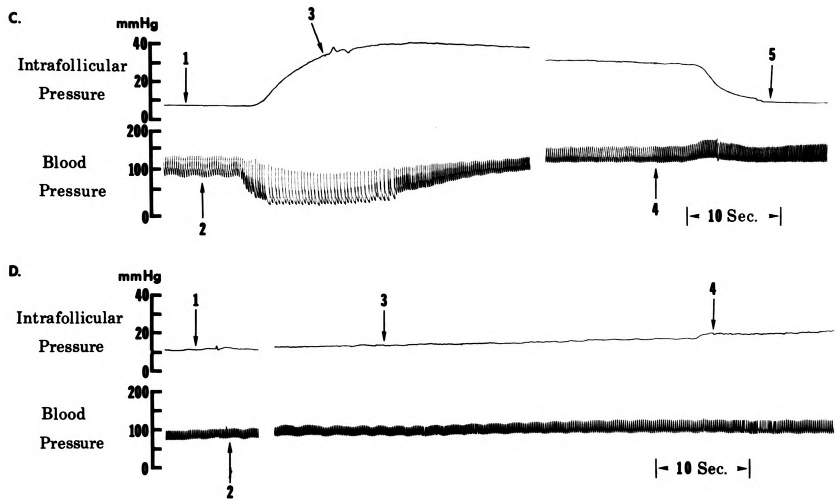
Acetylcholine is thought to be the natural mediator or stimulant between the post ganglionic cholinergic nerve endings and the receptors in smooth muscle. Acetylcholine is first liberated from nerve endings and then stimulates the appropriate muscle receptors. Acetylcholine esterase quickly hydrolyzes it. The receptors are stimulated by the depolarizing action of acetylcholine. Cutting (1964)

Figure 8-C. The effects of carbachol and atropine sulfate, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indicator of effect. The time break is equal to 25 seconds.

1. Feathers in loosened state.
2. Carbachol (cholinergic, 0.152 mg/kg) injected.
3. Feathers in tightened state.
4. Atropine (cholinergic blocking, 2.83 mg/kg) injected.
5. Feathers in loosened state.

Figure 8-D. The effects of physostigmine salicylate, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indication of effect. The time break is equal to 50 seconds.

1. Feathers in loosened state.
2. Physostigmine (cholinergic, 0.132 mg/kg) injected.
3. Feather pulling force increased, but not high enough to place the feathers in the tightened state.
4. Feathers in tightened state.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indicated that injected acetylcholine generally reaches only the post ganglionic receptors so that ganglionic and myoneural signs are minimal.

In pre-trial treatments, when using acetylcholine, the effects, as indicated by blood pressure, were rather short with no changes occurring in IFP and FPF. Acetylcholine esterase apparently inactivated acetylcholine rapidly. For this reason the birds were pre-treated with physostigmine which is a potent post ganglionic stimulant, after which acetylcholine was injected with a response being observed in IFP.

After the anesthetic, phenobarbital, was injected, IFP decreased to 42 percent of the original base pressure (example, Figure 7-A). Concurrent with the decrease in IFP, the feathers entered the loosened state and blood pressure decreased. After the injection of physostigmine (0.123 mg/kg), IFP gradually increased to 111 percent within 150 seconds. The feathers entered the tightened state in 143 seconds (example, Figure 8-D). During the same time period there was a slight increase in blood pressure.

When acetylcholine (0.466 mg/kg) was injected (189 seconds after physostigmine), IFP was at a 126 percent level with the feathers in the tightened state (example, Figure 9-E). IFP increased to a 258 percent level 10 seconds after the injection, while blood pressure and heart rate decreased. Immediately after this event, there was a

Figure 9-E. The effect of acetylcholine chloride and atropine sulfate, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with physostigmine salicylate. Blood pressure was recorded as an indicator of effect. The first time break is equal to 25 seconds; the second is equal to 124 seconds.

1. Feathers in tightened state.
2. Acetylcholine (cholinergic, 0.466 mg/kg) injected.
3. Atropine (cholinergic blocking, 2.83 mg/kg) injected.
4. Feathers in loosened state.

Figure 9-F. The effect of acetylcholine chloride, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with hexamethonium chloride and physostigmine salicylate. Blood pressure was recorded as an indicator of effect.

1. Feather pulling force increased, but not high enough to place the feathers in the tightened state. This section of record was taken 90 seconds after the injection of hexamethonium (ganglionic blocking, 7.3 mg/kg).
2. This section of record was taken 60 seconds after the injection of physostigmine (cholinergic, 0.132 mg/kg).
3. Approximately 206 seconds after the injection of physostigmine, feather pulling force had increased to a higher level, but not high enough to place the feathers in the tightened state.
4. Acetylcholine (cholinergic, 0.466 mg/kg) injected. The feathers entered the tightened state. The time break is equal to 40 seconds.

physostigmine (cholinergic, 0.137 mg/kg).

3. Approximately 206 seconds after the injection of physostigmine, feather pulling force had increased to a higher level, but not high enough to place the feathers in the tightened state.

4. Acetylcholine (cholinergic, 0.466 mg/kg) injected. The feathers entered the tightened state. The time break is equal to 40 seconds.

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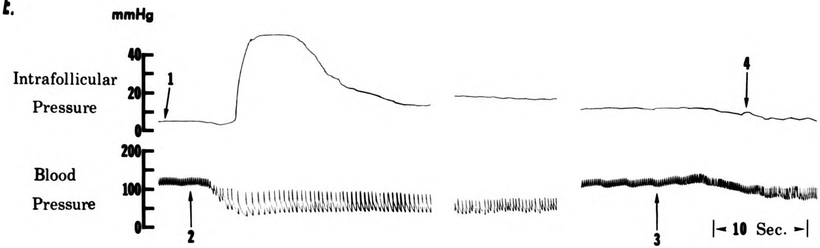

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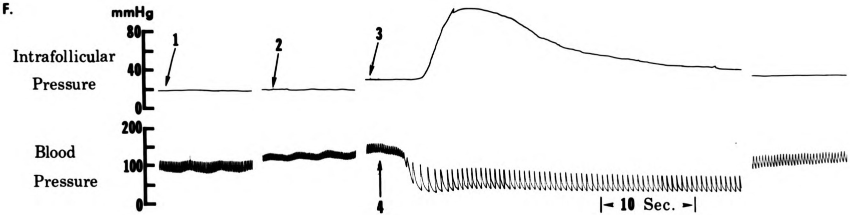


gradual decline in IFP and an increase in blood pressure and heart rate.

In birds treated with physostigmine, a potent cholinergic receptor stimulator, the IFP gradually increased and the feathers entered the tightened state. When acetylcholine was injected following physostigmine, there was a marked increase in IFP, while the feathers remained in the tightened state. These data indicate that the natural mediator between nerve endings and cholinergic receptors on smooth muscle, acetylcholine, caused IFP to change, thus producing a cholinergic receptor response in feather muscles.

Experiment 9.--Hexamethonium chloride and physostigmine salicylate pre-treatment followed by acetylcholine chloride (4 birds).

In the previous experiments, the cholinergic drugs which were used might have stimulated the autonomic ganglion to some extent thus causing an indirect stimulation of possible adrenergic receptors in the feather muscles. For this reason the birds in this experiment were pre-treated with a ganglionic blocking agent, hexamethonium. This agent acts by competing with acetylcholine for the ganglionic receptors (Cutting, 1964). Physostigmine and acetylcholine were again used to stimulate the cholinergic receptors of the feather muscles.

IFP decreased to 56 percent and the feathers entered the loosened state after the birds were anesthetized with

10 percent sodium phenobarbital (example, Figure 7-A). Blood pressure also concurrently decreased. IFP remained unchanged after hexamethonium (7.3 mg/kg) was injected, while FPF increased (example, Figure 9-F). The average FPF was 223 grams. This was slightly above that which was considered the loosened state (130 grams or less), yet well below the tightened state (500 grams or over). Blood pressure simultaneously decreased.

Physostigmine (0.132 mg/kg) was injected 197 seconds after hexamethonium. IFP increased to 67 percent within 20 seconds after injection and remained at this level through 150 seconds. FPF also increased to 302 grams, which was below that considered a tightened state for the feathers and blood pressure also increased.

At the time when acetylcholine (0.466 mg/kg) was injected, 210 seconds after the physostigmine injection, IFP had gradually increased to a 111 percent level. After acetylcholine was injected, IFP increased to a 389 percent level in 10 seconds, with a gradual decrease thereafter. The feathers concurrently entered the tightened state, while blood pressure and heart rate decreased.

When hexamethonium was injected, IFP remained unchanged, while the FPF increased to a level slightly above that which was considered as the loosened state for the feathers. Physostigmine was then injected and IFP increased slightly. FPF increased again, but not to the level that would have placed the feathers in the tightened state.

Finally, acetylcholine was given and IFP increased markedly, while the feathers entered the tightened state, thus indicating that the cholinergic receptors play an important role in feather release. In the previous experiment, physostigmine caused the feathers to enter the tightened state, but failed to do so in this experiment when the ganglion cells were blocked with hexamethonium. This might indicate that physostigmine was causing an increase in FPF through partial ganglionic stimulation.

Experiment 10.--Dichloroisproterenol and phenoxybenzamine pre-treatment followed by carbachol (3 birds).

There are two types of adrenergic receptors on smooth muscle. These receptors have been designated as alpha, which, when stimulated, causes vasoconstriction, and beta, which when stimulated, produces vasodilatation in arterioles (Drill, 1965). To eliminate possible interference from the stimulation of adrenergic receptors on the feather muscles, the birds in this experiment were pre-treated with phenoxybenzamine, an adrenergic alpha receptor blocker, and dichloroisoproterenol, an adrenergic beta receptor blocker. These adrenergic blocking agents exert their influence by combining directly in a competitive manner with adrenergic stimulating drugs for the adrenergic receptors. The birds were then given carbachol, a cholinergic drug.

Following the injection of an anesthetic, IFP decreased to 52 percent of the original base pressure (example, Figure 7-A). Concomitantly the feathers entered the loosened

state and blood pressure decreased. After dichloroiso-proterenol (6.5 mg/kg) was injected, IFP increased to a 157 percent level in 130 seconds (example, Figure 10-G). Blood pressure at first decreased and then increased, while the feathers remained in the loosened state.

One hundred and sixty (160) seconds later, phenoxy-benzamine (39.1 mg/kg) was injected, and IFP decreased to 33 percent in 90 seconds. The feathers remained in the loosened state, but blood pressure decreased.

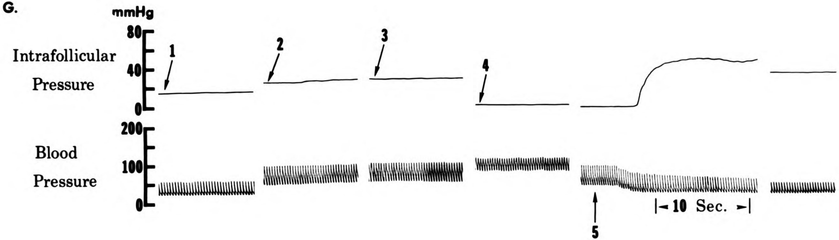
Carbachol (0.152 mg/kg) was injected 317 seconds after the injection of phenoxybenzamine. IFP increased rapidly to a 229 percent level within 6 seconds after injection. Sixteen (16) seconds later, IFP increased to a 343 percent level, which was followed by a gradual decline. The FPF increased to 181 grams which was slightly higher than that which was considered the loosened state and, yet, much lower than the tightened state. Blood pressure also concomitantly decreased. Two of the birds died from cardiac failure within 120 seconds after carbachol was injected.

Carbachol, a cholinergic receptor stimulating drug, caused an increase in IFP, but the feathers did not reach the tightened state in birds pre-treated with phenoxyben-zamine, an adrenergic alpha blocking agent, and dichloro-isoproterenol, an adrenergic beta blocking agent. Since two of the birds died with the final injection (carbachol), it is questionable as to whether or not the birds were

Figure 10-G. The effect of carbachol, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with dichloroisoproterenol and phenoxybenzamine. Blood pressure was recorded as an indication of effect.

1. Sixty (60) seconds after the injection of dichloroisoproterenol (adrenergic, beta blocking, 6.5 mg/kg).
2. One hundred and ten (110) seconds after the injection of dichloroisoproterenol. The feathers remained in the loosened state.
3. Ten (10) seconds after the injection of phenoxybenzamine (adrenergic, alpha blocking, 39.1 mg/kg).
4. Eighty (80) seconds after the injection of phenoxybenzamine. The feathers remained in the loosened state.
5. Carbachol (cholinergic, 0.152 mg/kg) injected. Feather pulling force increased, but was not high enough to place the feathers in the tightened state. The last segment on the graph was taken 60 seconds later and demonstrates the continued effects of carbachol.

G.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treated with too many drugs. It should be noted, however, that a cholinergic receptor response was obtained from the feather muscles, since IFP increased in birds with the adrenergic receptors blocked.

When examining the effects of the various cholinergic receptor stimulating drugs (pilocarpine, carbachol, physostigmine and acetylcholine) and the cholinergic receptor blocking drug (atropine) in Experiments 4 through 10, on IFP and FPF, the data indicate that the feather muscles have cholinergic receptors and that they play a major role in the feather tightening mechanism.

<u>The Effects of Adrenergic Drugs and Adrenergic Blocking Drugs on Intrafollicular Pressure and Feather Release</u>

Ostmann, <u>et al.</u> (1963b) noted that the feathers were loosened when birds were given an adrenergic blocking drug (yohimbine); however, these workers did not indicate whether the feather loosening was due to blocking of alpha or beta receptors. In the vascular system there are two types of smooth muscle receptors which respond in differing degrees to the various adrenergic drugs that mimic the effects of the sympathetic nervous system. Alpha receptors, when stimulated, cause vasoconstriction, while beta receptors cause vasodilatation. The following experiments were conducted to determine if feather muscles have adrenergic alpha and/or beta receptors.

Experiment 11.--Norepinephrine (6 birds).

Cutting (1964) indicated that norepinephrine is the principal product released when sympathetic nerves are stimulated and is generally liberated from the nerve endings near the effector sites. Norepineprine is principally an alpha receptor stimulator which causes contraction of smooth muscle in the vascular system. In this experiment, norepinephrine was injected to determine if the feather muscles have adrenergic receptors of the alpha type and if IFP and feather release would be altered.

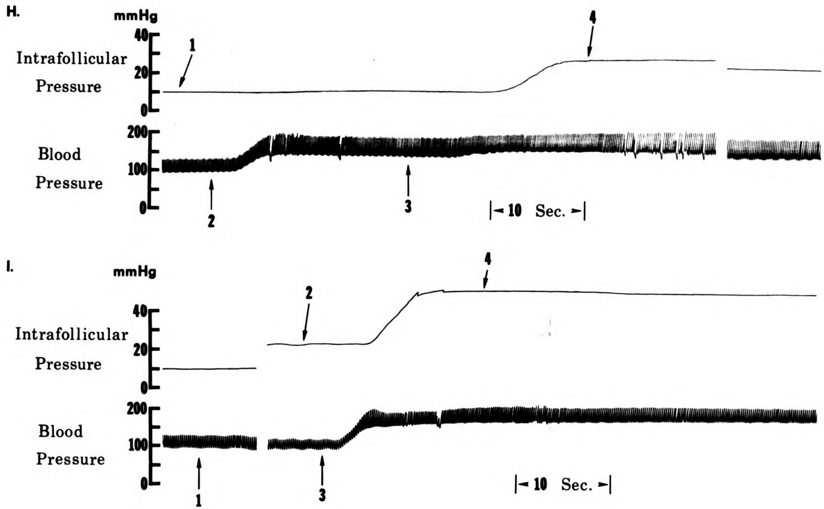
After the birds were anesthetized with 10 percent sodium phenobarbital, IFP decreased to 39 percent of the original base pressure (example, Figure 7-A). Blood pressure also decreased and the feathers entered the loosened state. Two (2) seconds after the injection of norepinephrine (0.112 mg/kg), blood pressure began to increase and reached its highest level in 16 seconds (example, Figure 11-H). IFP failed to respond appreciably. Approximately 20 seconds after the initial injection, a second dosage of norepinephrine (0.295 mg/kg) was injected, resulting in an increase in IFP beginning 8 seconds after the second injection. IFP increased from 45 percent to 148 percent in 18 seconds and thereafter gradually declined. Blood pressure remained constantly elevated from the first injection through the second injection and gradually diminished, concurrent with the decline in IFP. The feathers remained in the loosened

Figure 11-H. The effect of norepinephrine, when injected "to effect,"on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indicator of effect. The time break is equal to 30 seconds.

1. Feathers in loosened state.
2. Norepinephrine (adrenergic, 0.112 mg/kg)injected.
3. Second dosage of norepinephrine (0.295 mg/kg) injected.
4. Feathers remained in the loosened state.

Figure 11-I. The effect of norepinephrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with dichloroisoproterenol. Blood pressure was recorded as an indication of effect.

1. Dichloroisoproterenol (adrenergic, beta blocking, 6.5 mg/kg) injected.
2. Four (4) minutes after the injection of dichloroisoproterenol. The feathers remained in the loosened state.
3. Norepinephrine (adrenergic, 0.295 mg/kg) injected.
4. The feathers remained in the loosened state.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state throughout the experiment, that is, following the initial anesthetization.

When norepinephrine was injected, IFP increased, but the feathers remained in the loosened state. These data indicate that alpha receptors are present on feather muscles and are also involved in changing IFP, but have little effect of FPF.

Experiment 12.--Dichloroisoproterenol pre-treatment followed by norepinephrine (6 birds).

Since norepinephrine stimulates, primarily, alpha receptors, but also stimulates beta receptors to a slight degree, dichloroisoproterenol was used to block the beta receptors. Norepinephrine was then injected with only alpha receptors being stimulated and the effects on IFP and feather release were observed.

IFP decreased to 44 percent of the original base pressure after anesthetization with 10 percent sodium phenobarbital. Concomitantly the feathers entered the loosened state and blood pressure decreased (example, Figure 7-A). After dichloroisoproterenol (6.5 mg/kg) was injected, IFP gradually returned to the 100 percent level within 120 seconds (example, Figure 11-I). The feathers remained in the loosened state during this time period, while blood pressure flucturated, first decreasing and then increasing. Norepinephrine (0.375 mg/kg) was injected 174 seconds after the injection of dichloroisoproterenol. IFP then increased rapidly to a 206 percent level within 10 seconds, followed by a gradual increase to a 283 percent level in 22 seconds, and then gradually decreased. Blood pressure also increased markedly. The feathers remained in the loosened state throughout this experiment following initial anesthetization.

Again, as in experiment 11, but in this case with the beta receptors blocked, norepinephrine caused an increase in IFP, while the feathers remained in the loosened state. the data indicate that alpha receptors are present on feather muscles and influence IFP, but not FPF.

Experiment 13.--Epinephrine (2 birds).

Epinephrine was injected to further clarify the role played by the feather muscle alpha and/or beta receptors in feather release.

Epinephrine, an adrenergic drug, is liberated principally from the adrenal gland and stimulates both adrenergic alpha (pressor) receptors and adrenergic beta (dilator) receptors equally (Cutting, 1964). The effects of epinephrine were observed in IFP and feather release.

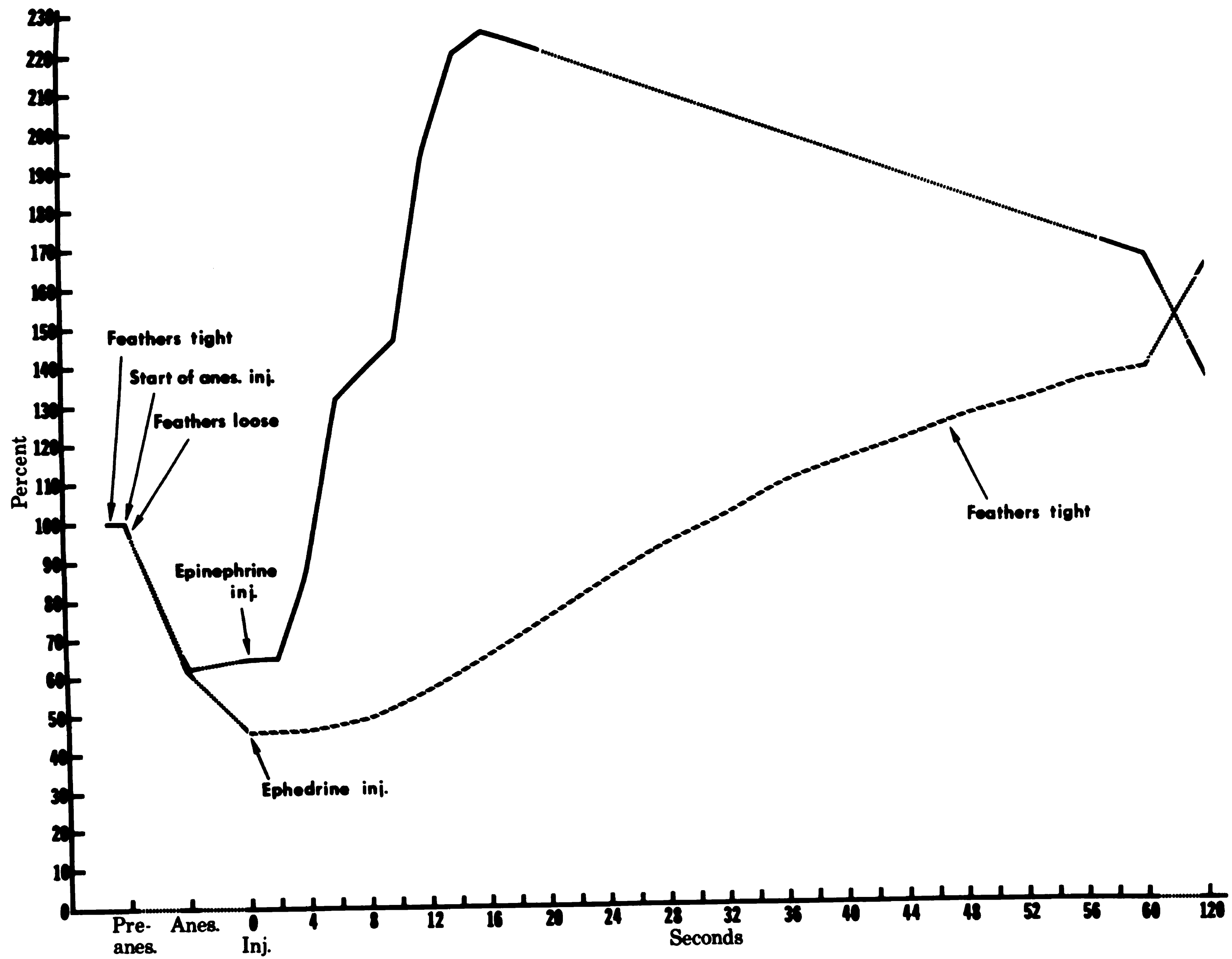
After anesthetization with 10 percent sodium phenobarbital, IFP decreased to 62 percent of the original preanesthetized base pressure (Figure 12). The FPF at this time also decreased and the loosened state.

After the injection of epinephrine (0.043 mg/kg), IFP increased rapidly from a 64 percent level to a 131 percent level in 8 seconds. Sixteen (16) seconds after the start of

Figure 12. The effects of ephedrine and epinephrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather pulling force (dorsal tract) of feather follicles abritrarily selected from S. C. White Leghorn hens. All percentages given are in relationship to intrafollicular pressure prior to anesthetization or drug treatment (100% as a base).

Ephedrine (adrenergic) (six birds used) 5.3 mg/kg ---------

Epinephrine (adrenergic) (two birds used) 0.043 mg/kg ——



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injection, IFP increased to its highest level (226 percent). At the termination of the experiment, 120 seconds after the start of injection, IFP had decreased to 136 percent. Since some of the feathers appared to be in the tightened state, while at the same time other feathers were in the loosened state, no conclusions were made as to whether or not the feathers were tight. Epinephrine did cause an increase in IFP.

The injection of epinephrine did not clarify the role played by the alpha and/or beta receptors in the feather release mechanism.

Experiment 14.--Epinephrine (7 birds).

This experiment is similar to Experiment 13, except that in addition to IFP and FPF, blood pressure was measured.

After anesthetization with 10 percent sodium phenobarbital, IFP decreased to 35 percent of the original base pressure (example, Figure 7-A). Concurrently, blood pressure and FPF also decreased.

After the injection of epinephine (0.043 mg/kg), IFP increased rapidly within 16 seconds to a level of 254 percent of the original base pressure (example, Figure 13-J). Approximately 4 seconds before the increase in IFP began, blood pressure started to respond and reached its highest level within 10 seconds of the epinephrine injection. After IFP reached its highest point, a slow decline followed. It was impossible to reach a conclusion on the effect of epinephrine upon the FPF due to the variation found.

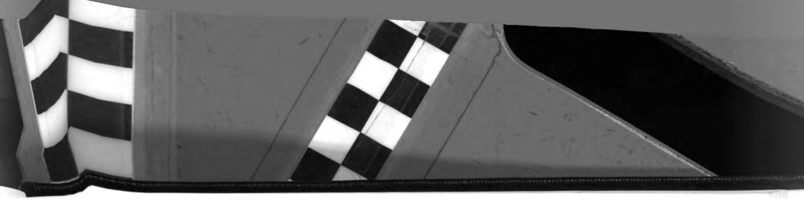
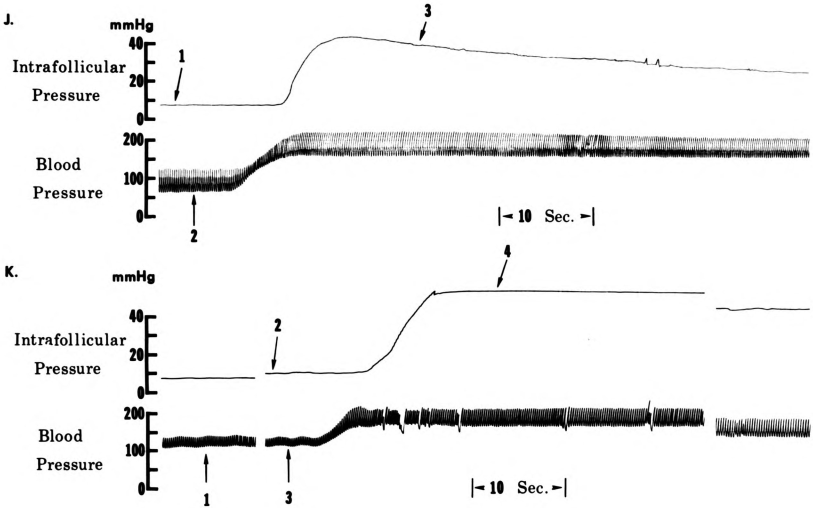


Figure 13-J. The effect of epinephrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indicator of effect.

1. Feathers in loosened state.
2. Epinephrine (adrenergic, 0.043 mg/kg) injected.
3. Feather pulling force was variable; some were in the loosened state while others were in the tightened state.

Figure 13-K. The effect of epinephrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with dichloroisoproterenol. Blood pressure was recorded as an indicator of effect.

1. Dichloroisoproterenol (adrenergic, beta blocking, 6.5 mg/kg) injected.
2. One hundred and seventy-eight (178) seconds after the injection of dichloroisoproterenol. The feathers remained in the loosened state.
3. Epinephrine (adrenergic, 0.043 mg/kg) injected.
4. The feathers remained in the loosened state. The time break is equal to 90 seconds.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|◄ 10 Sec. ►|

Epinephrine caused IFP to increase, but a large amount of variation was observed in FPF. Epinephrine stimulates alpha (pressor) receptors and beta (dilator) receptors equally (Drill, 1965). Since the data in this experiment did not clarify the role played by both the alpha and beta receptors, it is questionable as to whether both receptors are on feather muscles and if they are, as to how IFP and FPF might be affected by each.

Experiment 15.--Dichloroisoproterenol pre-treatment followed by epinephrine (6 birds).

Since epinephrine stimulates both alpha and beta receptors, dichloroisoproterenol was used to eliminate possible adrenergic beta receptor stimulation. Epinephrine was then used to stimulate the alpha receptors. The effects of alpha receptor stimulation were observed on IFP and FPF. IFP decreased to 61 percent of the original base pressure after anesthetization with sodium phenobarbital (example, Figure 7-A). Blood pressure also decreased and the feathers entered the loosened state.

After dichloroisoproterenol (6.5 mg/kg) was injected, IFP gradually increased to a 94 percent level in 110 seconds (example, Figure 13-K). Blood pressure also gradually increased during this period, while the feathers remained in the loosened state.

Epinephrine (0.043 mg/kg) was injected 180 seconds after dichloroisoproterenol. Within 16 seconds, IFP increased rapidly to a 228 percent level, with a gradual increase thereafter to a 272 percent level in an additional 8 seconds. This was followed by a gradual decrease. There was also a marked increase in blood pressure while the feathers remained loose during the treatment period.

Epinephrine, when injected into birds pre-treated with dichloroisoproterenol, a beta receptor blocker, caused IFP to increase, but the feathers remained loose. The data in this experiment indicate that alpha receptors on the feather muscles play a role in changing IFP, but have no influence on the feather release mechanism.

Experiment 16.--Atropine sulfate pre-treatment followed by epinephrine (2 birds).

Atropine was used in this experiment to block the possible effects cholinergic receptor stimulation. The birds were then injected with epinephrine to stimulate the alpha and/or beta receptors and the effects were observed on IFP and FPF.

After the birds were anesthetized IFP decreased to 50 percent of the original base pressure (example, Figure 7-A). Simultaneously the feathers entered the loosened state and blood pressure decreased.

IFP, FPF and blood pressure remained unchanged after the injection of atropine (2.83 mg/kg, example, Figure 14-L).

When epinephrine (0.043 mg/kg) was injected, 230 seconds later, IFP increased to a 406 percent level within 18 seconds and then gradually decreased. The feathers remained in the

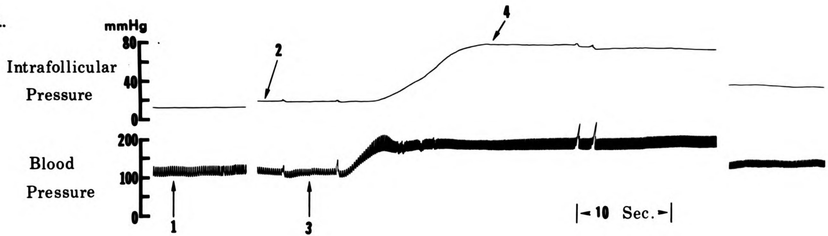
Figure 14-L. The effect of epinephrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with atropine sulfate. Blood pressure was recorded as an indicator of effect.

1. Atropine (cholinergic blocking, 2.83 mg/kg) injected.
2. Four (4) minutes after the injection of atropine. Feathers remained in the loosened state.
3. Epinephrine (adrenergic, 0.043 mg/kg) injected.
4. The feathers remained within the loosened state. The time break is equal to 90 seconds.

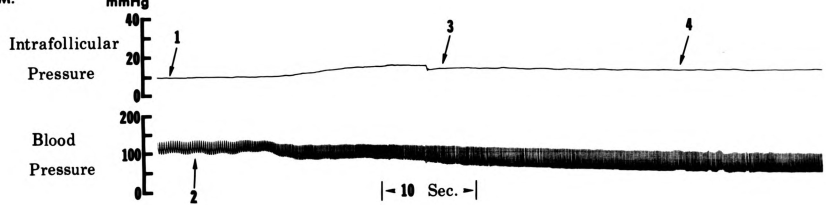
Figure 14-M. The effect of isoproterenol hydrochloride, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen.

1. Feathers in the loosened state.
2. Isoproterenol (adrenergic, beta stimulating, 1.02 mg/kg) injected.
3. Feather pulling force increased but was not high enough to place the feathers in the tightened state.
4. Feathers in the tightened state.

L.


mmHg
80
40
0
Intrafollicular
Pressure
200
100
0
Blood
Pressure
1
2
3
4
|◄ 10 Sec. ►|

M.


mmHg
40
20
0
Intrafollicular
Pressure
200
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0
Blood
Pressure
1
2
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4
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loosened state throughout this treatment period, while blood pressure increased markedly.

When epinephrine was injected into birds pre-treated with atropine, IFP increased, but the feathers remained in the loosened state. Why some of the feathers tightened to different degrees when epinephrine was given by itself is not understood. Based on the data from this experiment and Experiment 15, possibly the cholinergic or the adrenergic beta receptors might have caused the partial feather tightening as in Experiment 14 where the birds were treated with only epinephrine. No feather tightening occurred when the cholinergic and adrenergic beta receptors were blocked; while, on the other hand, epinephrine, when stimulating alpha receptors, caused IFP to increase in these experiments.

Experiment 17.--Isoproterenol (6 birds).

Isopreterenol stimulates the adrenergic beta receptors in the smooth muscle of the arterioles in skeletal muscles causing the blood vessels to dilate. Since adrenergic alpha receptor stimulation alone failed to cause feather tightening, the possibility of adrenergic beta receptors influencing IFP and feather release was investigated.

IFP decreased to 62 percent of the original base pressure after anesthetization (example, Figure 7-A). The feathers entered the loosened state, while blood pressure decreased.

IFP increased to a 110 percent level within 22 seconds after isoproterenol (1.02 mg/kg) was injected (example, Figure 14-M). By 28 seconds after injection, IFP had decreased to a 95 percent level and remained at this level for the remainder of the experiment. Blood pressure decreased and tachycardia occurred. The feathers gradually entered the tightened state within 43 seconds after injection.

When isoproterenol was given, the vascular response was immediate, while IFP increased slowly and the feathers gradually entered the tightened state. Why the feather muscles did not respond immediately is not known. Probably there are no beta receptors on feather muscles and the slow reaction might have been due to an ischemic condition causing stimulation of the other receptors or the muscles themselves, since the birds became quite pale in appearance upon drug treatment.

Experiment 18.--Phenoxybenzamine pre-treatment followed by isoproterenol (3 birds).

Phenoxybenzamine, an adrenergic alpha blocking agent, was injected as a pre-treatment to eliminate any possible adrenergic alpha receptor effects when isoproterenol was injected. IFP, FPF and blood pressure were recorded.

IFP decreased to 48 percent of the original base pressure following anesthetization with 10 percent phenobarbital (example, Figure 7-A). Blood pressure also decreased and the feathers entered the loosened state.

After phenoxybenzamine (39.1 mg/kg) was injected, IFP decreased to a 14 percent level within 70 seconds, while the feathers remained in the loosened state (example, Figure 15-N). A depression of blood pressure and tachycardia were exhibited.

One hundred and ninety-three (193) seconds later, isoproterenol (1.02 mg/kg) was injected. IFP did not change and the feathers remained in their loosened state for the duration of this treatment (160 seconds). Concurrently blood pressure decreased and tachycardia occurred.

These data indicate that isoproterenol may have had an indirect effect on IFP and FPF in Experiment 17, since, when the alpha receptors were blocked with phenoxybenzamine, IFP did not change and the feathers remained in the loosened state. If beta receptors are present on feather muscles, it appears that they do not play an important role in changing IFP or in feather release.

Experiment 19.--Ephedrine (6 birds).

Ephedrine is a synthetic drug which exhibits most of the effects of epinephrine by stimulating the same adrenergic receptors. This drug is less potent, but has a longer action than does epinephrine. Ephedrine also exhibits a moderate amount of central stimulation, much more than that of epinephrine (Cutting, 1964). The effects of alpha and beta receptor stimulation by this drug were observed on IFP and feather release.

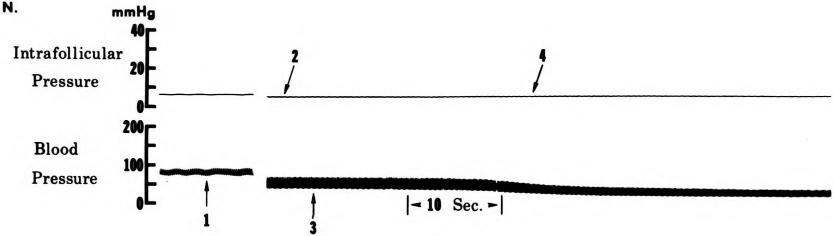
Figure 15-N. The effect of isoproterenol, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen pre-treated with phenoxybenzamine. Blood pressure was recorded as an indicator of effect.

1. Phenoxybenzamine (adrenergic, alpha blocking, 39.1 mg/kg) injected.
2. One hundred and sixty (160) seconds after phenoxybenzamine was injected. The feathers remained in the loosened state.
3. Isoproterenol (adrenergic, beta stimulating, 1.02 mg/kg) injected.
4. Feathers remained in loosened state.

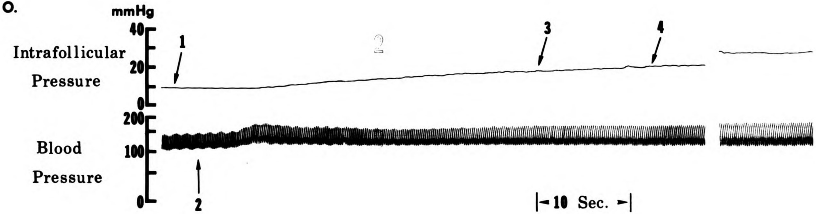
Figure 15-O. The effect of ephedrine, when injected "to effect," on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from an anesthetized S. C. White Leghorn hen. Blood pressure was recorded as an indicator of effect.

1. Feathers in loosened state.
2. Ephedrine (adrenergic, 5.3 mg/kg) injected.
3. Feather pulling force increased but was not high enough to place the feathers in the tightened state.
4. Feathers in tightened state.

N.


mmHg
40
20
0
Intrafollicular
Pressure
200
100
0
Blood
Pressure
1
2
3
4
|◄ 10 Sec. ►|

O.


mmHg
40
20
0
Intrafollicular
Pressure
200
100
0
Blood
Pressure
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|◄ 10 Sec. ►|

After the birds were anesthetized with 10 percent sodium phenobarbital, IFP decreased to 45 percent of the original base pressure, while FPF decreased.

Immediately after the injection of ephedrine (5.3 mg/kg), IFP increased in a gradual and steady rate reaching a level of 138 percent of the original base pressure in 60 seconds. In an average of 46 seconds after the start of injection, the feathers entered the tightened state. At this time, IFP had attained a level of 123 percent. At the termination of the experiment, 120 seconds after the start of drug injection, IFP had increased to 164 percent of the original base pressure.

Ephedrine, when injected, caused a gradual increase in IFP and the feathers gradually entered the tightened state. This drug reacted more like isoproterenol (beta stimulating) than epinephrine, in that the feathers gradually entered the tightened state. Thus, the use of ephedrine did not clarify the role of the alpha and beta receptors in the feather tightening mechanism.

Experiment 20.--Ephedrine (6 birds).

This experiment was the same as Experiment 19, but with blood pressure measurement included. After the birds were anesthetized, IFP decreased to 47 percent of the original base pressure. Concurrent with the drop in IFP, blood pressure and the FPF decreased (example, Figure 7-A). After the infusion of ephedrine (5.3 mg/kg), IFP slowly increased reaching a level of 139 percent of the original base pressure in 158 seconds (example, Figure 15-0). The feathers reached the tightened state in 82 seconds, while blood pressure increased rapidly within 10 seconds and remained at this elevation throughout the remainder of the experiment.

Again, as in Experiment 19, ephedrine, when injected, caused a gradual increase in IFP and the feathers gradually entered the tightened state. This drug did not clarify the role played by the alpha and beta receptors in the feather release mechanism.

In Experiments 11 through 20, the stimulation of the isolated alpha receptors on feather muscles caused a rapid and marked increase in IFP, while the feathers remained in the loosened state. When a beta receptor stimulating drug (isoproterenol) and ephedrine, which stimulates beta receptors equally as well as alpha receptors, were given, a gradual increase in IFP occurred and the feathers gradually entered the tightened state. Since alpha receptor blockage nullified the effects of beta receptor stimulation on feather release, it is questionable as to whether beta receptors are present on feather muscles.

The Effects of Neuromimetic Drugs on Feather Shaft Movement

In the previous experiments it was noted that when the various adrenergic and cholinergic drugs were injected, there were feather shaft movements associated with changes in IFP.

The effects of some of the above mentioned neuromimetic drugs upon the movements of the feather shafts of the femoral feather tract in the living bird were observed. These observations were made through a nine power magnification binocular stereoscope focusing on clipped or trimmed feather shafts before, during and after injection of the drugs.

Experiment 21.--To determine if the alpha receptors on the feather muscles effect feather shaft movement, a total of 7 birds were injected with epinephrine (0.043 mg/kg).

In each case, the feathers were observed to have a marked movement toward the epithelial surface (drawn tight to the body) and were held in this position until the effects of the drug subsided. The feathers eventually returned to their normal position (partially erected) in relation to the skin. The FPF following the treatment with epinephrine presented a varied picture as seen in previous experiments, with some feathers in the tightened state and some not. In the previous experiment (17), in which isoproterenol (adrenergic, beta receptor stimulating) was used, little or no feather shaft movement was observed.

When the alpha receptors on feather muscles are apparently stimulated, IFP increases and simulataneously the feather shafts become drawn tight to the body.

Experiment 22.--The following experiment was undertaken to observe the effects of cholinergic receptor stimulation on feather shaft movement.

Preliminary observations on the effects of cholinergic drugs, pilocarpine nitrate (5.6 mg/kg, 5 birds) and carbachol (0.152 mg/kg, 3 birds), when followed by treatment with a cholinergic blocking agent (atropine sulfate, 2.83 mg/kg), on feather shaft movements were difficult to observe due to the fact that the respiratory movements were so prominent and therefore obscured shaft movements. With this treatment, the feather shaft movements were slight in comparison with those seen in epinephrine treated birds. In an effort to overcome the problem produced by the respiratory movements, the 5 birds reported on were anesthetized with 3 percent sodium pentobarbital to the point at which respiration ceased. The birds were then given artificial unidirectional respiration (Burger and Lorenz, 1960). An injection of pilocarpine (5.6 mg/kg) was then given and the feather shafts were observed to twist and elevate slightly. The FPF also entered the tightened state. These data indicate that cholinergic receptors play a role in feather shaft movement, as well as IFP changes and the feather tightening mechanism.

When alpha receptors are apparently stimulated on feather muscles, the feather shafts are drawn tight to the body, while on the other hand, when cholinergic receptors are stimulated, the feathers become slightly elevated.

DISCUSSION

Ostmann, et al. (1963a) indicated that there are no muscles within the follicular wall and that the feather muscles between follicles insert into each individual follicle via elastic fibers from elastic tendons. In an untreated control bird, one might conceive that the nervous system continuously stimulates the feather muscles; thus, tension from the feather muscles acts upon the elastic fibers within the follicular wall in such a manner as to produce pressure on the feather shaft and thereby produce the tightened state within the feather follicle. Therefore, a certain amount of muscle contraction would be required before the elastic fibers within the follicular wall could produce enough tension to cause pressure on the feather shaft, thereby increasing feather pulling force (FPF) and placing the feathers in the tightened state. In the case of certain drugs, such as anesthetics and neuromimetic blocking agents, which cause the feathers to loosen (Ostmann, et al., 1963b), one might conclude that the feather muscles relax, thus reducing tension on the elastic and also the collagen fibers within the wall of the feather follicle.

To study the feather release mechanism, a technique was developed to measure the pressure within the feather follicle.

After death, intrafollicular pressure (IFP) and FPF are apparently not related. As soon as the FPF increased to a level at which the feathers were considered in the tightened state, it remained at this level regardless of the IFP level. It should also be remembered that when the nervous system is depressed or blocked, the feathers enter the loosened state (Ostmann, et al., 1963b); while, on the other hand, after death the nervous system is no doubt inoperational. This presents the possibility of different mechanisms producing and maintaining the tightened state, before and after death, within the feather follicle. Probably, the feather tightening mechanism is influenced for one or two hours after death by local factors, such as the feather muscles being stimulated by a possible change in pH and by using energy stored in the muscle for contraction in an anaerobic environment vs. an aerobic state when alive.

In birds killed by cervical dislocation, the feathers entered the tightened state much more rapidly than did the feathers in birds killed with an overdose of anesthetic. The mass spasmodic contraction of feather muscles observed in the first case was probably responsible for initiating the tightened state within the feather follicle much faster than that initiated in birds killed with an overdose of anesthetic as muscle spasms were not observed in the latter case.

When the birds were anesthetized, it was noted that the feathers entered the loosened state almost immediately, yet IFP decreased at a much slower rate. On the other hand, when a drug was given that would cause the feathers to enter the tightened state, it was noted that this event occurred at a time when IFP was well above the original 100 percent base pressure.

The difference in time observed between the IFP increase and the onset of the tightened state could be explained by the fact that the cannulated follicle was filled with fluid vs. a solid feather shaft in the natural state. In this case, the feather muscles would be working against a liquid filled follicle which could change shape, while on the other hand, in the natural state, the follicle is filled with a solid shaft which probably does not change shape when tension is placed on or in the follicle.

No doubt, one of the main functions of the feather muscles is that of changing the position of the feather shafts as observed in Experiments 21 and 22. If this is a predominant factor, the changes observed in IFP are probably related to this function, thus obscuring any minute pressure changes which might be involved between the loose and tightened state of the feather follicle.

One of the above stated reasons might account for the lag observed between the increase or decrease in IFP and the feathers entering or leaving the tightened state.

The drugs discussed below were given to determine whether or not the receptors of nervous innervation on the feather muscles were of a cholinergic and/or adrenergic (alpha and/or beta) nature. The effects of these drugs on IFP and feather release was also studied.

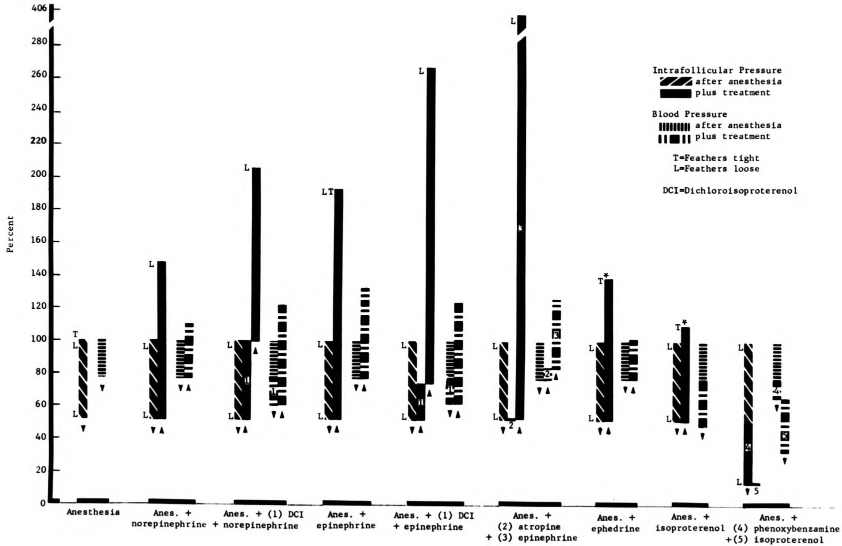
Drill (1965) indicated that there are two types of adrenergic receptors on the smooth muscle in the walls of the blood vessels in mammals. The receptors designated as alpha cause vasoconstriction when stimulated, while the so-called beta receptors generally found in the walls of blood vessels in skeletal muscle cause vasodilatation. According to Drill (1965), the adrenergic class of drugs usually stimulates both of these smooth muscle receptors, but to differing degrees. Norepinephrine stimulates mainly alpha receptors; epinephrine and ephedrine stimulate both alpha and beta receptors equally, while isoproterenol stimulates chiefly beta receptors. The stimulation of these two adrenergic receptors and their effects on the vascular system of the chicken, which were found to be similar to that reported for the mammal, were reported by Harvey, et al. (1954) and Bunag and Walaszek (1962). To determine if feather muscles have alpha and/or beta receptors and to determine the effects of these receptors on IFP and feather release, the sequence of drugs discussed below was used.

When several adrenergic drugs (Figure 16) were given, varied results, insofar as their effectiveness in causing the feathers to enter the tightened state were obtained. Norepinephrine, according to Cutting (1964) and Drill (1965), was shown to be secreted by the post ganglionic sympathetic nerve endings and to principally stimulate the alpha receptors on smooth muscle. When this particular adrenergic drug was injected into an anesthetized chicken, IFP increased immediately and the feather shafts were depressed to the body in the area of the femoral feather tract. Norepinephrine did not cause the feathers to enter the tightened state. Thus, it appears that there are alpha receptors on the feather muscles and that they are involved in changes in IFP and feather shaft position.

Epinephrine, an adrenergic drug which according to Drill (1965) stimulates both alpha and beta adrenergic receptors equally, when injected into the anesthetized chicken, produced the same effects as norepinephrine with one exception. Some of the feathers entered the tightened state, to varying degrees, while others did not. When the beta receptors were blocked and epinephrine was then injected, the feathers remained in the loosened state; thus, again indicating that alpha receptors are on feather muscles and that they are involved in changes in IFP and feather shaft movement but are not a factor in the feather tightening mechanism.

When ephedrine, a synthetic adrenergic drug with similar effects as from epinephrine, was injected, blood pressure increased immediately, as it did with the two

Figure 16. A summation of the effects of several adrenergic and adrenergic blocking drugs when injected "to effect" on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from S. C. White Leghorn hens. Blood pressure was recorded as an indicator of effect. *When these two drugs were given intrafollicular pressure gradually increased in an average of 149 seconds versus 4 seconds for the other drugs described.



Intrafollicular Pressure
after anesthesia
plus treatment
Blood Pressure
after anesthesia
plus treatment
T=Feathers tight
L=Feathers loose
DCI=Dichloroisoproterenol
Percent
406
280
260
240
220
200
180
160
140
120
100
80
60
40
20
0
Anesthesia
Anes. + norepinephrine
Anes. + (1) DCI + norepinephrine
Anes. + epinephrine
Anes. + (1) DCI + epinephrine
Anes. + (2) atropine + (3) epinephrine
Anes. + ephedrine
Anes. + isoproterenol
Anes. + (4) phenoxybenzamine +(5) isoproterenol

previously mentioned drugs, while on the other hand, IFP increased gradually. The feathers also entered the tightened state gradually. Cutting (1964) stated that ephedrine is a strong central stimulator; epinephrine also is, but to a lesser extent. This might account for the different degrees of feather tightening observed when using the above mentioned adrenergic drugs.

Wood, et al. (1963), in the dog and Harvey, et al. (1954) and Bunag and Walaszek (1962), in the chicken, demonstrated the reversal effects of epinephrine in the systemic system. As previously stated, in the vascular system, epinephrine can stimulate both alpha (vasoconstrictor) and beta (vasodilator) receptors of the smooth muscle within the blood vessel walls. This system allows for both higher systemic blood pressure and a greater flow of blood through exercising muscles. Abboud, et al. (1965) measured the effect of beta adrenergic stimulation on the small blood vessels of muscle in the foreleg of the dog using blood pressure measurements and found that vasodilatation occurred.

Since alpha receptors were shown to be present on feather muscles, the question arises as to whether the smooth feather muscles have beta receptors. When isoproterenol (beta stimulating) was injected, IFP increased slowly and the feathers gradually entered the tightened state. No marked feather shaft movement was observed and blood pressure decreased. The birds in the subsequent experiment were pre-treated with phenoxybenzamine (alpha blocking) after which isoproterenol was injected. In this case, IFP remained unchanged, the feathers stayed in the loosened state and blood pressure decreased.

When the birds were treated with isoproterenol, the comb and skin became quite pale in appearance possibly due to a shift in blood flow to the skeletal muscles, thus secondary effects set in, such as a shift in metabolism, resulting in the stimulation of other receptors or of the feather muscles themselves causing the feathers to enter the tightened state. This shift in blood flow might have caused ischemia or changed the metabolism from an aerobic to an anaerobic state in the feather muscles, thus causing the muscles to react in a similar manner as when the birds were killed (Experiments 1 through 3) which resulted in the feathers entering the tightened state. Somewhat analogous observations were made by Daniell and Bagwell (1966) who reported that isoproterenol stimulation caused acute changes in myocardial metabolism. Lactate utilization and pH were found to drop in isoproterenol treated cardiac muscle, also a decrease in coronary blood flow was noted.

In the birds treated with cholinergic drugs (Figure 17), carbachol, pilocarpine, physostigmine, and acetylcholine, IFP increased, the feathers entered the tightened state and, in the femoral feather tract, the feather shafts became erect. These results indicate that there are cholinergic receptors on feather muscles and that they play a role in changing IFP, FPF and in changing feather shaft

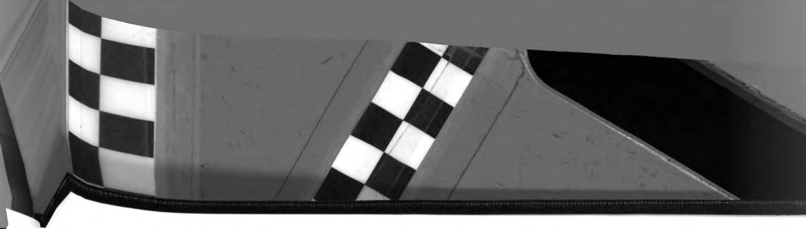
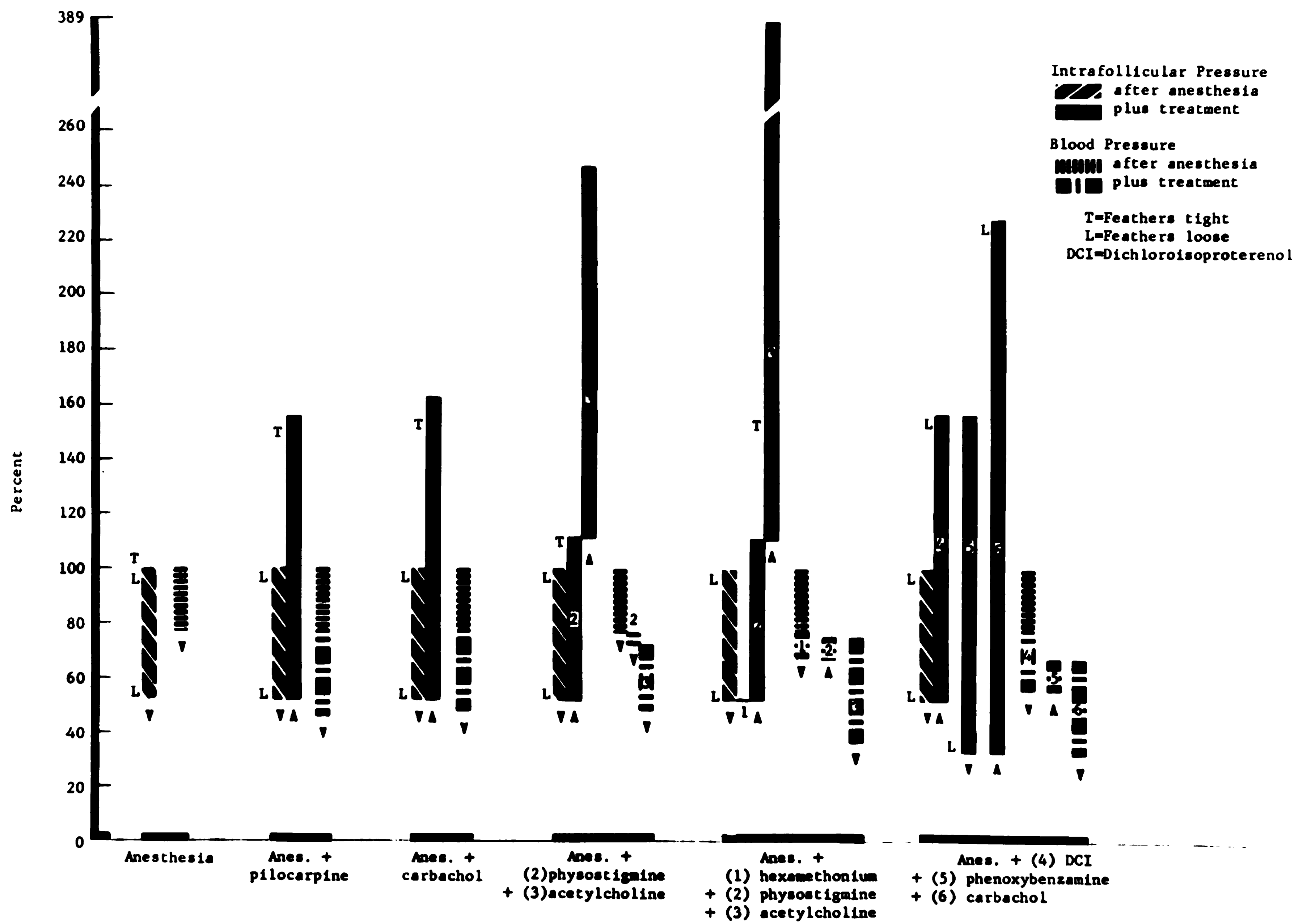


Figure 17. A summation of the effects of several cholinergic drugs when injected "to effect" on intrafollicular pressure (femoral tract) and feather release (dorsal tract) of feather follicles arbitrarily selected from S. C. White Leghorn hens. Blood pressure was recorded as an indicator of effect.

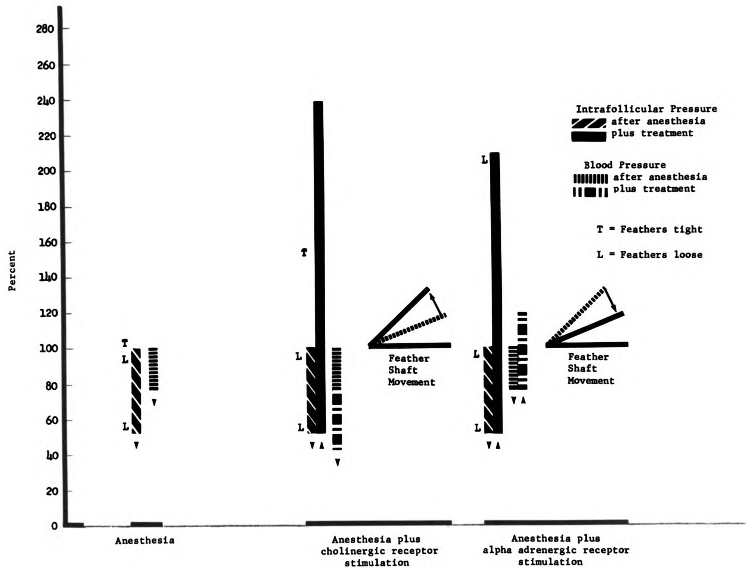

Percent
389
260
240
220
200
180
160
140
120
100
80
60
40
20
0
Anesthesia
Anes. + pilocarpine
Anes. + carbachol
Anes. + (2)physostigmine + (3)acetylcholine
Anes. + (1) hexamethonium + (2) physostigmine + (3) acetylcholine
Anes. + (4) DCI + (5) phenoxybenzamine + (6) carbachol
Intrafollicular Pressure
after anesthesia
plus treatment
Blood Pressure
after anesthesia
plus treatment
T=Feathers tight
L=Feathers loose
DCI=Dichloroisoproterenol

position. Since acetylcholine is rapidly destroyed by cholinesterase (Drill, 1965), the birds were pre-treated with physostigmine, which, besides being a strong cholinergic stimulator, acts as an inhibitor of this enzyme. To eliminate the effects of adrenergic receptors, one group of birds was pre-treated with both alpha (phenoxybenzamine) and beta (dichloroisoproterenol) adrenergic blocking agents and then injected with carbachol (cholinergic receptor stimulating), the result being an increase in IFP. Blood pressure was markedly decreased after carbachol was injected, with two out of the three birds dying in less than 120 seconds. However, the FPF did not increase. This was probably due to the fact that the systemic system was greatly depressed.

To eliminate the possibility that the cholinergic drugs used were stimulating the post ganglionic sympathetic fibers, several birds were pre-treated with hexamethonium, a ganglionic blocking agent. In addition, these birds were pre-treated with physostigmine and then injected with acetylcholine resulting in an increase in IFP. Also, the feathers entered the tightened state, thus eliminating ganglionic stimulation as a possible factor in feather tightening.

These data seem to indicate that, in the chicken, the feather muscles have both cholinergic and alpha adrenergic receptors (Figure 18) which are arranged on the feather muscles of the femoral feather tract in such a manner as to

Figure 18. A summation of the effects of cholinergic and alpha adrenergic receptor stimulating drugs on intrafollicular pressure (femoral tract), feather release (dorsal tract) and feather shaft movement (femoral tract) of feather follicles arbitrarily selected from S. C. White Leghorn hens. Blood pressure was recorded as an indicator of effect.


Percent
280
260
240
220
200
180
160
140
120
100
80
60
40
20
0
T
L
L
T
L
L
L
L
L
Feather
Shaft
Movement
Feather
Shaft
Movement
Anesthesia
Anesthesia plus
cholinergic receptor
stimulation
Anesthesia plus
alpha adrenergic receptor
stimulation
Intrafollicular Pressure
after anesthesia
plus treatment
Blood Pressure
after anesthesia
plus treatment
T = Feathers tight
L = Feathers loose

cause feather shaft erection when the feather muscles are stimulated by cholinergic drugs, while adrenergic drugs cause the feather shafts to become depressed. This differs from mammals where only cholinergic receptors located on the pilomotor muscles and which are innervated by only the sympathetic nervous system caused erection of the hair shaft (Ruch and Fulton, 1960).

Cholinergic receptor stimulation caused the feathers to enter the tightened state while alpha adrenergic receptors when stimulated had no effect in producing the tightened state within the feather follicle.

SUMMARY AND CONCLUSIONS

An experiment was conducted to determine the mechanism(s) involved in the loose and tightened states within the feather follicle by measurement of intrafollicular pressure (IFP). When birds were anesthetized, IFP decreased, also, simultaneously, the feather pulling force (FPF) decreased. After death, the FPF and IFP were apparently not related. Once the FPF had increased to where the feathers were considered tight, it remained there regardless of the IFP level.

In the anesthetized chicken, stimulation of alpha receptors on feather muscle resulted in an increase in IFP, while the feather tightening mechanism remained in the loosened state. The feathers remained in the loosened state when norepinephrine was injected, varied between loose and tight with the injection of epinephrine and gradually entered the tightened state with ephedrine. An adrenergic beta stimulating drug (isoproterenol) caused the feathers to enter the tightened state. However, when the birds were pre-treated with an alpha blocking (phenoxybenzamine) drug and the beta receptors then stimulated, the feathers remained in the loosened state. This may indicate that in the first case, secondary factors were involved in stimulating receptors other than beta receptors. Adrenergic alpha stimulating drugs caused the feather shafts in the femoral feather tract to become depressed to the body.

Cholinergic receptor stimulating drugs (pilocarpine, carbachol, physostigmine and acetylcholine) caused an increase in IFP, erection of the feather shafts, decreased blood pressure and also, the feathers to enter the tightened state; thus, indicating the presence of cholinergic receptors on the feather muscles.

In comparing the effects of the various drugs used in the anesthetized birds and their effects upon the feather tightening mechanism, it appears that the cholinergic receptors when stimulated produced the tightened state within the feather follicle, while the adrenergic alpha receptors when stimulated did not cause feather tightening, although both receptors caused IFP to increase and the feather shafts to change position.

The feather muscles appear to have both adrenergic alpha and cholinergic receptors as compared to only cholinergic receptors on the pilomotor muscle of the hair follicle in mammals.

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