THE PHYSIOLOGY OF THE HUMAN AXILLARY APOCRINE SWEAT GLAND*†

WALTER B. SHELLEY, M.D., PH.D. AND HARRY J. HURLEY, JR., M.D.

For over two centuries anatomists have known that there are large sweat glands in the axillae of man. These apocrine glands have been carefully studied by histologists and pathologists, yet only scant attention has been given them by the physiologist (1). The need for further knowledge of the physiology of these organs is apparent to the dermatologist, and recently Lobitz and Campbell (2) have undertaken a physiological study of the apocrine glands of the human ear canal. Concurrently, we are studying the axillary apocrine glands in a similar manner (3, 4). The present account details some of the physiological data obtained.

METHOD

In this investigation observations were largely confined to gross direct visualization of apocrine sweating. The axillae served as the test site since apocrine sweat glands are most abundant in this area. Normal human volunteers served as subjects. The tests were made during all seasons of the year. The data was derived from study of men who either had not used axillary deodorants or antiperspirants for two weeks or not at all. The observations on women are limited since it was difficult to secure subjects who would or had avoided axillary preparations.

The axilla was shaved using either a safety razor or an electric clipper. The individual's arm was kept extended laterally to keep the axillary vault exposed during the entire course of observations. Although visualization of the apocrine sweat droplet could be achieved with the unaided eye, observations were always made with an otoscope or a dissecting microscope (15×). Specimens were collected by means of fine glass capillary pipettes, either placed directly on the skin surface or inserted into the hair follicle (Fig. 1). Fluorescence was determined by the use of a standard Wood Light. The pH was determined by the use of Universal indicator papers.‡ Pharmacologic agents were prepared in sterile physiologic saline solution in concentrations ranging from 1/1,000 to 1/100,000. In some instances a stock commercial epinephrine solution was used. In others, a buffered saline solution (Sorenson phosphate buffer, pH 7.4) was used to minimize local pain during injection. Heat stress was produced by having the subjects remain in an infra-red heat cabinet (5) for periods up to one hour.

* This study was supported by Contract DA-49-007-MD-154 with the Medical Research and Development Board, Office of the Surgeon General, Department of the Army.

† Department of Dermatology (Donald M. Pillsbury, M.D., Director), University of Pennsylvania Medical School, Philadelphia, Pennsylvania.

Presented at the Thirteenth Annual Meeting of The Society for Investigative Dermatology, Inc., June 7, 1952, Chicago, Illinois.

‡ pHydrion papers, Micro Essential Laboratory, Brooklyn, New York.

RESULTS

Apocrine Sweat

The apocrine sweat was seen to appear commonly at the hair follicle, although in most subjects it also appeared at sites independent of hair follicles.

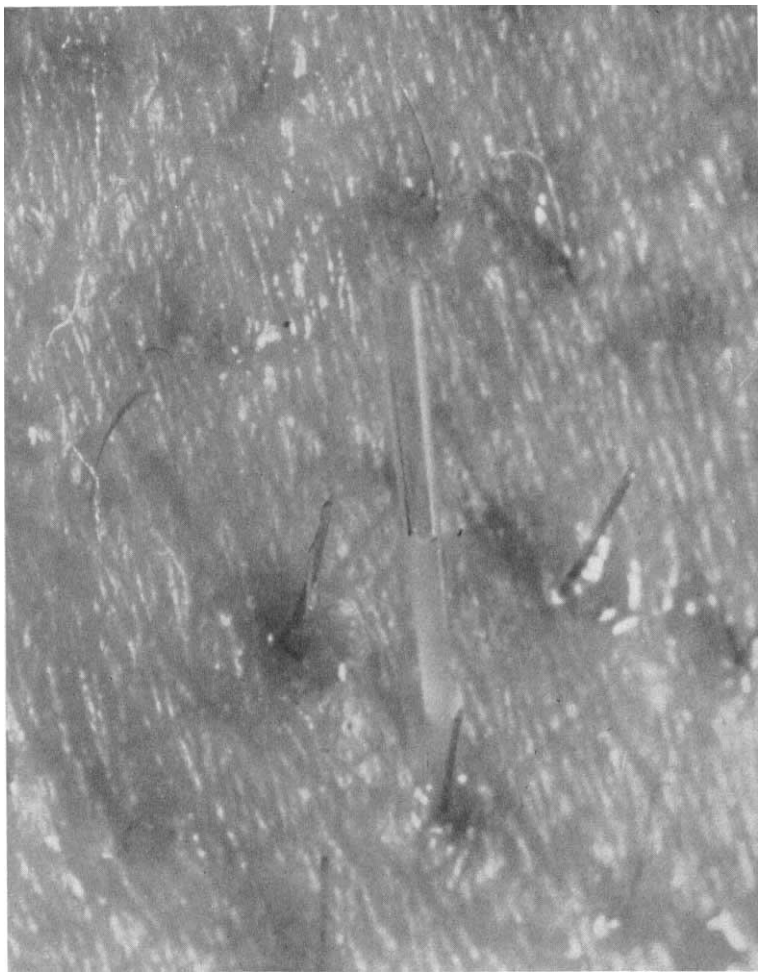

FIG. 1. Apocrine Sweat in Axilla

Dried apocrine sweat appears as glistening caps about hair follicles. Observe capillary tube in hair follicle opening containing whitish apocrine sweat.

Actually, in some subjects a relatively large number of the apocrine glands opened independently onto the skin surface. Apocrine sweat was identified as a whitish or greyish fluid (Fig. 1), occasionally showing a definite yellowish tint. It usually did not form a spherical droplet such as eccrine sweat commonly does. The droplet was translucent but not transparent. The milky or

soapy appearance showed great individual variation. In some subjects the color was so faint that identification could be made only by comparative appearance in the capillary tubes, held against a black background. In others the secretion appeared more of the consistency and appearance of thick cream. In some instances the apocrine sweat spreads over the perifollicular skin in a thin film, never forming a droplet. This made identification very difficult. The total quantity produced was minute when compared to the volume of eccrine sweat which may be seen. A reasonable estimate of the quantity of apocrine sweat produced by an individual gland in response to stimulation is 0.001 ml. Thus, the total quantity of apocrine sweat which could be produced was only a small fraction of a milliliter.

If not diluted by eccrine sweat the apocrine sweat droplets dried to form a glistening glue-like mass. This appeared as a yellowish somewhat adherent cap over the hair follicle or the apocrine orifice (Fig. 1).

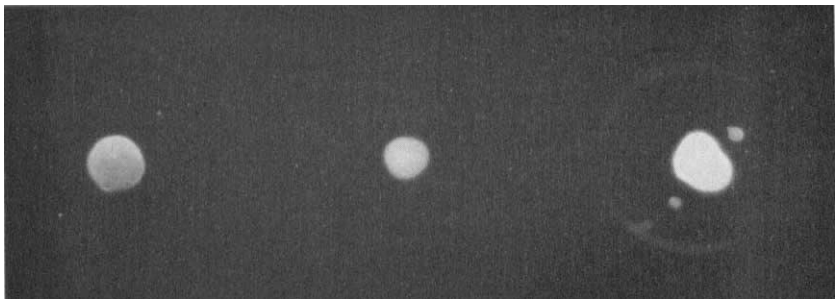

FIG. 2. Apocrine Sweat Fluorescence. Three droplets of dried apocrine sweat are shown under Wood Light.

In most of the subjects studied, the apocrine sweat was fluorescent (Fig. 2). Eccrine and apocrine sweat could be sharply distinguished on the basis of this fluorescence. It was impossible to detect an odor to these minute quantities of apocrine sweat in most individuals, although in some subjects a definite odor appeared at the time of apocrine sweating.

In studying the pH of apocrine sweat, sixteen normal adult male subjects were used. Micro-samples of uncontaminated apocrine and eccrine sweat droplets were obtained from the axillae. The apocrine sweat was produced by local epinephrine injections, whereas the eccrine was emotional in origin. pH measurements were made using universal pH paper with which 0.5 pH unit changes can be detected. Comparative studies revealed the pH of apocrine sweat to range between 5.0 and 6.5, whereas eccrine sweat ranged between 4.0 and 6.0. As a rule the apocrine sweat appeared to have a pH approximately an 0.5 unit higher than that of eccrine sweat, but the differences were too slender to permit identification of the type of fluid simply on pH measurement.

The size of apocrine sweat samples necessitated the employment of ultra-micro-analytic technics. Spot tests (6) proved to be the most satisfactory method

of determining the chemical composition of this fluid. It was found that apocrine sweat contains protein (using a α-nitrose β-naphthol test for tyrosine), carbohydrate (reducing sugars using silver nitrate), ferric ion (α,α' dipyridyl after reduction), and ammonia (manganese nitrate, silver nitrate).

Effect of Emotions

Apocrine sweating was seen in response to such stresses as evoked pain and fear. Observations on patients seen on routine clinic visits revealed that in the occasional patient apocrine sweating could be seen. This was more common in individuals apprehensive concerning the possibility of skin cancer, or in individuals about to have minor dermatologic surgery. It was far less common than the usual emotional eccrine sweating seen under these circumstances. The amount of eccrine sweating and apocrine response could not be correlated.

Seventeen patients were carefully studied prior to being subjected to a lumbar puncture. In only one of these individuals was apocrine sweating seen in the axilla. This is similar to one series of observations on consecutive clinic patients in whom 2 out of 15 showed apocrine sweating.

Another study of the effect of apprehension or fear was conducted on 33 experimental subjects. These men were entirely unaware of what experimental procedures might be conducted, and many of them showed distinct apprehension. However, only 5 of the 33 showed apocrine sweating prior to the testing.

The effect of pain was also studied. It was found that rather severe pain was necessary to produce apocrine sweat. In the series of observations on the 17 patients having a lumbar puncture, 3 of these individuals showed apocrine sweating immediately following pain during the puncture. The application of an electrically heated wire to the skin led to the production of apocrine sweating in 2 of 11 subjects studied. Radiant heat was also applied to the skin of 2 subjects. In both of these, severe pain was produced, and apocrine sweating resulted. In 25 subjects a No. 18 needle was thrust into the palm to produce pain. In 10 of these individuals apocrine sweating resulted.

The effects of pain on apocrine sweating could best be studied in women during labor. It was found that mild uterine contractions were not associated with apocrine response. However, intense contractions producing severe pain were associated with definite apocrine sweating. Within a few seconds after the onset of each intense labor pain, 6 to 10 apocrine sweat droplets appeared. Observations over a period of an hour indicated that each burst of apocrine sweating involved new glands. Secretion did not reappear at former sites of activity.

It was found possible to block the apocrine sweat gland response to pain by the introduction of procain into the skin. Sixteen subjects received an injection of 4% aqueous procaine intradermally. Five to ten minutes later after local anesthesia had been produced, the 16 subjects received a painful stimulus consisting of the introduction of a needle into the palmar skin. In 4 of these, apocrine sweating was seen in the unanesthetized control areas. In none of these subjects was apocrine sweating seen in the anesthetized areas.

The apocrine response to pain was further studied by the use of a localized external pressure which obliterated the circulatory supply. A pressure of 225 mm. of mercury was applied to a small area of the axillary skin of 14 subjects using a glass cup device, with an attached sphygmomanometer. Control observations had shown that the pressure did not produce apocrine sweating, or did it in any way inhibit an apocrine response. The 14 subjects were given painful stimulus consisting of the introduction of a needle into the palm. In 5 of the 14 individuals, apocrine sweat was noted in the normal control areas (outside the pressure zone). In 4 of the 14 subjects, apocrine sweat was noted simultaneously in the pressurized area. This apocrine sweat response to pain appeared within one second and was generalized throughout the axillary region unlike that to be described below seen in response to drugs.

Effect of Heat

Fifteen subjects were placed in an infra-red heat cabinet for periods of approximately thirty minutes. True apocrine sweating was observed in only one of these subjects as a result of this heat stimulus, although a marked out-pouring of eccrine sweating was seen in all subjects. It is interesting to note in this connection that most of the subjects showed follicular eccrine sweating. Capillary tubes were placed in the follicular orifices in 5 of the subjects, and it was impossible to demonstrate the secretion of any apocrine sweat. The one subject in whom apocrine sweat was observed during heat showed definite signs of anxiety and apprehension, and it was assumed that his apocrine sweating was on the basis of an emotional stimulus rather than the thermal one.

Effect of Drugs

Sympathomimetic Drugs:

Epinephrine in a 1:1,000 concentration in sterile saline solution was injected intradermally in 10 subjects. The quantities injected varied from .01 ml. to 0.2 ml. In each of the 10 subjects, apocrine sweating was seen to appear at the follicular orifice or at random points. The larger quantities produced stimulation of more glands since there was a greater spread of the drug. There was a minimal latent period of 15 seconds. Usually the secretion appeared directly in the injection area about a half minute after the introduction of the epinephrine. This secretion continued for a period of from 5 to 10 minutes with an ever widening zone in which glands began to show activity. The active glands were almost invariably located in the zone showing blanching from the epinephrine.

It was possible also to produce apocrine sweating by the systemic administration of epinephrine. In these instances it was necessary to give rather large doses of the epinephrine. Apocrine sweating was not seen until systemic signs of the epinephrine also appeared. It could usually be produced by the injection of 1 ml. of 1:1,000 solution of epinephrine.

The local introduction of *nor-epinephrine* (levophen bitartrate, 0.05 ml. of 1:1,000 solution) in 7 subjects also produced apocrine sweating in all of them.

The injection of epinephrine into atropinized areas in 2 subjects led to apocrine sweating. In 16 subjects a small area was anesthetized by the local introduction of 1 cc. of 4% aqueous solution of procaine. Epinephrine given into the normal unanesthetized areas produced apocrine sweat in all 16. In the anesthetized areas apocrine sweating was produced in 15 of the 16 subjects.

Epinephrine in 1:100,000 dilution also produced apocrine sweating.

Para-Sympathomimetic Drugs:

Acetyl choline (.05 ml. of 1:1,000 aqueous solution freshly prepared) was introduced intradermally in the axilla of 10 subjects. In none of these was true apocrine sweating observed. Again, however, eccrine follicular sweating was noted.

Pilocarpine (.05 ml. of 1:1,000 sterile buffered saline) was introduced intradermally in the axilla of 25 subjects. In 3 of these true apocrine sweating was seen. In 12 of them clear eccrine follicular sweating was observed. Normal eccrine sweating was seen in all 25 subjects.

Miscellaneous:

Control observations on the saline solution in 7 subjects resulted in no apocrine sweating. The injection of atropine solution (.05 ml. of 1:1,000 solution) in 3 subjects was without effect. In 16 subjects the injection of 4% aqueous solution of procaine led to localized apocrine sweating in 3 individuals. It was found that any painful cutaneous injection in the axilla might cause apocrine sweat to appear.

Effect of External Pressure

Using a glass cup device attached to an aneroid sphygmomanometer by means of a side arm, it was possible to subject the localized area of the axilla to high external pressure. In 5 subjects it was found that the application of this pressure did not stimulate any apocrine sweat gland activity. The following experiment was then conducted: Epinephrine (.05 ml. of 1:1,000) was injected intradermally. Within one to two seconds a pressure of 225 plus or minus 10 mm. of mercury was applied to the injection site. The pressure was maintained for 3 minutes. During this period apocrine sweating was observed in the pressurized area, and no apocrine sweat was seen in the surrounding zone. However, within a minute after removal of the pressure, apocrine sweating could be seen to appear in the surrounding area. This was presumably due to the lymphatic spread of the epinephrine which had been previously prevented by the pressure of the rim of the cup.

Effect of Age

Eleven children from the age of 5 to 10 years were studied. This group consisted of 3 negro boys, 3 negro girls, 3 white boys, and 2 white girls. Each was given .05 ml. of 1:1,000 solution of epinephrine in the axilla. In none of them was apocrine sweating seen. A pain stimulus was also given to each of the children consisting of the introduction of a hypodermic needle, No. 25, into the

skin of the forearm. Again, no apocrine sweating was seen. It should be pointed out that none of these children had hair in the axilla, other than the lanugo type.

Thirty-five normal, healthy adult males ranging in age from 15 to 50 years were also studied. Each of this group was given a painful stimulus by means of the introduction of a No. 18 needle into the palm. Fourteen of the individuals showed apocrine sweating. These subjects were also studied after .15 ml. of 1:1,000 solution of epinephrine was introduced into the axilla. Definite apocrine sweat activity was seen in each of the 35 subjects. However, the degree of response showed marked individual variation.

A number of studies were also made, with similar findings, on the apocrine response in female subjects. However, the data on the women are not included since most of them had used antiperspirants extensively. It has been assumed that these antiperspirants may reduce or obliterate the apocrine sweat gland response (7).

An older age group was also studied. This consisted of 11 male subjects ranging in age from 60 to 75 years. After experiencing the pain of the palmar injection, in 3 of the 11 men apocrine sweating could be noted. Each of them also received an injection of 0.1 ml. of 1:1,000 solution of epinephrine in the axilla. Six of the 11 subjects showed an apocrine gland response to this.

Refractory Period

A study of the individual apocrine gland led to the finding that there was a definite refractory period. It was found that after an initial injection of epinephrine it was not possible to produce sweat from any unit which had responded, until a period of hours had elapsed. We have not determined the exact duration of this refractory period, but can state that it exceeds 24 hours in the case of epinephrine stimulation.

Miscellaneous Observations

It was found that the apocrine glands in the pubic areas and about the nipple responded to both the pain stimulus and to epinephrine.

The apocrine gland did not respond to the local cooling of the skin produced by the application of ice cubes for a period of 3 minutes. The injection of methylene blue into the skin and subsequent injections of epinephrine did not lead to the production of blue-colored apocrine sweat.

No spontaneous apocrine sweating was observed in subjects at rest. One individual had capillary pipettes placed in the follicular orifices, and during a period of 3 hours no fluid or follicular material could be seen to enter these pipettes, nor was any dried apocrine sweat seen on the skin surface.

Apocrine sweat never appeared at all hair follicle orifices in response to a stimulus. In one such study, 63 hair follicles were observed following local epinephrine. Sixteen glands were activated, 14 of these being follicular, whereas 2 were non-follicular.

Discussion

At the beginning of this study it became apparent that there are two distinct types of sweat which may appear at the hair follicle orifice in response to various stimuli. It was found that the follicular sweating may be either clear or turbid. On the basis of anatomic and cytologic study, it can be stated that the turbid fluid appearing in the hair follicle is apocrine sweat. It is identical with the turbid sweat seen to appear at random points other than the hair follicle. The clear, colorless follicular sweat thus could be either apocrine or eccrine. In other words, if we define apocrine sweat as any sweat appearing in the follicular orifices, we are forced to assume that the apocrine sweat gland produces two entirely different types of secretion. We have elected, however, to consider that the apocrine sweat gland produces only one type of secretion, namely turbid lactescent fluid. We feel that the clear fluid appearing at the hair follicle is eccrine sweat. We have developed this view as a result of consideration of the following points:

(1) The physical appearance of this follicular sweat is identical with that of ordinary eccrine sweat. It appears to us more reasonable to assume that this sweat has been produced by the eccrine gland rather than the apocrine gland, since these two glands each have a homogenous type of secretory epithelium.

(2) Although anatomically we can find no eccrine sweat ducts emptying into the hair follicle, we have observed them opening commonly at the lip of the follicular orifice. It would seem that eccrine sweat would flow out of the orifice and down into the hair follicle, giving a false impression as to the site of origin. A study of hairy areas of the body in which no apocrine glands exist revealed that eccrine sweat may appear to arise within the hair follicle.

(3) Pharmacologically the clear fluid is seen to develop in response to all of the stimuli which activated the eccrine sweat gland. In other words, we have seen this clear follicular sweat arise in response to acetyl choline, pilocarpine, and also as a result of thermal stimulation. If one were to assume that this apocrine gland were producing this clear fluid, it would be necessary to assume that the gland was under the control of both adrenergic and cholinergic fibers. This does not appear likely.

(4) From the histochemical standpoint, the apocrine and eccrine glands can be distinguished on the basis of their glycogen content. The eccrine gland which produces clear fluid, has large supplies of glycogen, in contrast to the absence of glycogen in the apocrine gland. It would seem that if the apocrine gland were to produce large quantities of clear sweat, one might expect to find glycogen within the secretory cell.

Although we have proceeded with the fact that the apocrine sweat gland produces only one type of secretion, the intriguing possibility that a second type of secretion might occur is still under further study.

The possibilities for error in the identification of apocrine sweat are mani-

fold. One may confuse follicular eccrine sweat with apocrine sweat if identification is based solely on the source of the sweat. Also, eccrine sweat may be mistaken for apocrine sweat if the eccrine sweat is contaminated by soap, powders, or antiperspirant-deodorant preparations. Many individuals may show profuse, turbid sweat dripping out of the axilla during physical examinations. This is usually not apocrine sweat, but rather contaminated eccrine sweat.

It is also possible to conclude erroneously that no apocrine sweat is present. The apocrine sweat is readily diluted by eccrine sweat, removing all identifiable characteristics. At other times no droplet of apocrine sweat forms or the quantity is visible only under magnification.

Turning to the nature of apocrine sweat, it is important to point out that the quantities of this sweat are extremely minute. They in no way compare with the relatively large amounts of eccrine sweat which appear. The size of specimens precluded the possibility of other than ultramicrochemical analyses. Furthermore, pH measurements had to be made using indicator paper.

Probably the fluorescence of the apocrine sweat is related to the content of solid material. We have no indication as to the identity of the fluorochrome present in apocrine sweat. There would seem to be a definite difficulty in the further study of this since again the question of size of sample is a critical one.

The literature has long cited odor as a distinctive sign of the presence of apocrine sweat. Our study would lend to the belief that this factor may have been over-emphasized. There are several other significant sources of odor in the axilla, and we feel that it is precarious to base any conclusions on the activity of the apocrine sweat gland on the basis of olfactometric observations.

As a result of the physiologic data presented above, we have postulated that the apocrine sweat gland is under the control of the autonomic nervous system. From the pharmacologic standpoint it is served by the adrenergic fibers. There do not appear to be any inhibitory fibers leading to the gland.

The following features characterize the control of the gland:

(1) It responds to sympathomimetic drugs and not to para-sympathomimetic agents.
(2) It shows instantaneous response to strong emotional stimuli, such as pain and fear.
(3) Its response to emotional stimuli is blocked by the local infiltration of anesthetics.
(4) Its activity is not blocked by the local arrest of the circulation.

The apocrine gland in man stands in marked contrast to the eccrine gland. It does not respond to acetyl choline, pilocarpine, or to thermal stimulation. Moreover, it is not nearly so readily responsive to stimuli as the eccrine gland. It always requires a relatively strong neural stimulus to activate it. It was seen that the pain of mild uterine contractions was insufficient to stimulate the apocrine gland, whereas the eccrine gland responded well to these. Again, in many subjects the painful stimulus of the injection of fluid into the palmar skin did not lead to any activity of the apocrine gland, whereas the eccrine

gland showed marked response. This is in consonance with the observations of Lobitz who found that it was difficult to stimulate the apocrine sweat gland of the external auditory canal (2).

The physiologic observations on the effect of age are confirmatory evidence for the views held by the histologists. The apocrine gland does not develop until puberty, and thus it is not surprising to find an absence of apocrine sweat in the younger age group. Again in the post-climacteric group the gland shows the twilight of its function. Histologically it becomes atrophic. However, it is of interest to note that true apocrine sweating was observed in an individual seventy-five years of age.

The apocrine gland shows a true refractory period, both to neural as well as drug stimulation. This period of inactivity lasts for hours and probably is associated with a period of secretion into the lumen. Correlative cytologic and physiologic data are now being obtained concerning this point.

It is tempting to theorize regarding the mechanism of apocrine gland function. On the basis of our present knowledge, one might assume that the apocrine gland cells form sweat by a process of true secretion. This sweat may then be stored in the gland until adrenergic stimuli cause its expulsion by means of myoepithelial contraction. This latter phenomenon of expulsion would be perceived by the observer as apocrine sweating, yet it may be distinct from the true osmotic cellular process of secretion of sweat. Further study is necessary before any conclusions can be drawn. For the present we have felt it advisable to realize that there may well be a distinction between *apocrine sweat secretion* and *apocrine sweating*. This is analogous to the mammary gland wherein the secretion of milk and lactation are two separate processes.

SUMMARY

1. Apocrine sweating could be stimulated by emotional stimuli of significant intensity.

2. Apocrine sweating was produced by epinephrine and nor-epinephrine.

3. It was not possible to produce apocrine sweating as a result of acetyl choline, pilocarpine or thermal stimulation.

4. The apocrine secretion was seen to appear against an external pressure of 225 mm. of mercury.

5. Apocrine sweating could not be produced in children, but was seen in adults. There was a reduction in the quantity of apocrine sweat in the older age group.

6. The apocrine sweat gland unit showed a definite refractory period, both to neural as well as chemical stimulation.

7. It has been postulated that apocrine sweating is under the control of adrenergic fibers of the autonomic nervous system.

Acknowledgments

We are indebted to Dr. William F. Morrison, Miss Sondra Golomb and Mr. Walter Davis for assistance in the prosecution of part of this work. Our

thanks is due also to Mr. Robert Morris for recording the fluorescence of apocrine sweat.

REFERENCES

1. Woolard, H. H.: The cutaneous glands of man. J. Anat., **64:** 415, 1930.
2. Lobitz, W. C. and Campbell, C. J.: Physiology of the glands of the human ear canal: Preliminary report apparatus and technique. J. Invest. Dermat., **19:** 125, 1952.
3. Shelley, W. B.: Apocrine sweat. J. Invest. Dermat., **17:** 255, 1951.
4. Shelley, W. B. and Hurley, H. J.: Methods of exploring human apocrine sweat gland physiology. Arch. Dermat. & Syph., **66:** 156, 1952.
5. Shelley, W. B., Horvath, P. N., Weidman, F. D. and Pillsbury, D. M.: Experimental miliaria in man: I. Production of sweat retention anidrosis and vesicles by means of iontophoresis. J. Invest. Dermat., **11:** 275, 1948.
6. Feigl, F.: Qualitative Analysis by Spot Tests. 3rd Edition. Elsevier Publishing Company, Inc., New York, 1947.
7. Sulzberger, M. B., Zak, F. G. and Herrmann, F.: Studies of sweating: II. On the mechanism of action of local antiperspirants. Arch. Dermat. & Syph., **60:** 404, 1949.

DISCUSSION

Dr. Stephen Rothman, *Chicago, Ill.:* I was impressed by this beautiful presentation. I was rather surprised to hear that no clear-cut difference has been found between the pH's of eccrine and apocrine secretions. Uniformly, all previous investigators found eccrine sweat definitely more acid than apocrine sweat. Also, I wonder how it can be proved that if apocrine glands respond to heat, this is not a thermal but an emotional response. I do not say that the statement is incorrect but I do believe the authors have not proved that apocrine glands do not respond to heat stimuli. According to Kuno apocrine responses to thermal and mental stimuli can be well distinguished.

Dr. Theodore Cornbleet, *Chicago, Ill.:* We, too, like many others found apocrine sweat more alkaline than eccrine. The latter averages about pH 4.5, while the former is about 6.5. It seems the study of the apocrines is coming into its own belatedly. It seems strange that these structures, which bear such a close relationship to that of the breasts, should have been so neglected. In cystic mastitis, the breast glands show a strong resemblance to the apocrines. We should take advantage of the fact that these structures are easy to get at and ready indicators of hormonal effects. Some of the specimens we studied seemed to show that the myoepithelium could act as an expulsion organ. There appears to be a break in these structures in hidradenitis as cause or effect, according to our studies.

Dr. Franz Herrmann, *New York, N. Y.:* The most stimulating study of Shelley and Hurley gives rise to various new questions. I agree with the two previous discussers who recalled the fact that the pH of apocrine sweat is generally regarded as higher than that of eccrine sweat (Arch. für Dermat. und Syph. **158:** 290, 1929; Dermat. Wchnschr. **88:** 397, 1929; Journ. Invest. Dermat. **7:**

215, 1946). As Thurmon and Ottenstein (Journ. Invest. Dermat. **18:** 333, 1952) have recently pointed out, this difference in pH is paralleled by a difference in the ammonia concentration which is substantially higher in sweat from the axillae than in sweat from areas with only eccrine glands. The described absence of apocrine sweating in response to heat or to the administration of cholinergic drugs seems to be at variance with the observations of several other investigators (Arch. für Dermat. und Syph. **158:** 290, 1929; Arch. Dermat. and Syph. **49:** 410, 1944; Journ. Invest. Dermat. **7:** 215, 1946; ibid. **18:** 71, 1952). In a previous discussion (Symposium on Sweating, Bronx. Dermat. Soc., Dec. 20, 1951, In Press, Arch. Dermat. and Syph.), I already had occasion to mention most of the—perhaps only apparently—conflicting points. In a recent publication Wada (Science **114:** 123, 1951) demonstrated cholinergic, as well as thermal sweating in the dog, in addition to adrenergic sweating. This is in agreement with various earlier descriptions of physiologic sweating upon thermal or cholinergic stimulation in other animals which likewise possess only apocrine sweat glands. Thus, one would have to assume principally different autonomic mechanisms in all these animals on one hand, and in man on the other. We have the impression that it is difficult, if not impossible, to afford direct evidence of an absence of apocrine sweating in response to heat or cholinergic stimulation, since dilution of the apocrine excretion by eccrine sweat would be unavoidable.

We observed relatively larger quantities of thermal sweat in the axillae of females than in those of males (Journ. Invest. Dermat. **18:** 71, 1952), inasmuch as the amounts in this site were similar in both sexes, whereas the males showed greater quantities in practically all other sites; and we explained this observation by the large apocrine apparatus in females. It appears very unlikely that our result was produced by emotion. In most of our volunteers (medical students) the exposures to heat were frequently repeated, without major differences in the results; the degree of heat was relatively moderate (dry bulb temperature averaged about 108°F, wet bulb temperature about 78°F). Drs. Shelley and Hurley have emphasized today that only profound emotional stress is capable of stimulating the "apocrine function". It is hardly conceivable that such stress could have occurred under our experimental conditions.

I still feel that the dual function of the apocrine glands,—i.e. their ability to produce sweat similar to the liquid delivered by the eccrine glands, as well as viscous material containing disintegrated epithelium—and a dual responsiveness to different autonomic stimuli may furnish an explanation of the different forms of reactivity observed.

Dr. Harry J. Hurley, Jr., *Philadelphia, Penn. (in closing):* I certainly appreciate these very stimulating comments and should like to thank Drs. Rothman, Cornbleet, and Herrmann for their interest.

Universal indicator paper was used in our pH studies and we collected pure samples of eccrine and apocrine sweat from the axillae. Only very slight differences were noted, if any, and although the apocrine sweat tended to be less

acid, this minimal variation could hardly be exploited practically in identification of the secretion, since only a 0.5 pH unit difference was seen. On another body site, perhaps greater variation would be apparent. We intend to investigate this further.

Dr. Cornbleet spoke of the myoepithelium and its possible function. This structure has been under investigation for many years and is found in other organs also, including mammary and eccrine glands. To it has been ascribed the role of expulsion of the apocrine sweat, but as yet we are unable to definitely prove its function. Theoretically, the myoepithelium might contract progressively along the tubule in a manner analagous to a peristaltic wave, to result in the expulsion of already formed apocrine sweat. This is an appealing explanation of its action. However, the fact that apocrine sweat appears against an external pressure of 225 mm Hg would not seem to support such a concept. In addition, preliminary observations of *in vitro* studies do not reveal any myoepithelial contraction.

Dr. Hermann suggests that the apocrine gland could be responsible for two different types of secretion. This is an exciting possibility but we have little evidence that would permit us to concur.

Clear follicular fluid may be seen to appear in response to pilocarpine or heat, but we feel this fluid to be identical with true eccrine sweat and not an additional type of secretion from the apocrine gland. While eccrine ducts do not discharge into the hair follicle itself, they can be seen to dump into the lip of the follicle or so close to it as to collect there and so give one the false impression of origin from within the follicle. Moreover, it is improbable that a homogeneous secretory cell type epithelium could be responsible for two types of secretion.

Finally, in only a very small percentage of cases did we see true, turbid apocrine sweat in response to pilocarpine, and it was felt this was probably the result of local pain.