(*1–3*). However, leucocytosis and thrombocytosis following epinephrine have been observed in splenectomized patients (*4*), and it is doubtful that the bone marrow is capable of immediately delivering such large numbers of leucocytes and platelets into the circulation. As part of an investigation of the hematological role of the lung, 0.1–0.2 mg of epinephrine was administered intravenously to several patients with metastatic neoplastic diseases. By frequent sampling of blood from intravascular catheters placed in the right ventricle and an appropriate large artery, it was observed that the increase in number of leucocytes and platelets in the arterial samples preceded and exceeded that found in the venous blood by at least one to two circulation times (Fig. 1). The arterial-venous platelet difference was more marked and sustained than the leucocyte difference.

It would thus appear that the pulmonary circulation in man may act as an available source of leucocytes and platelets, which may be delivered rapidly into the peripheral circulation under the stimulus of intravenous epinephrine administration. The lung, therefore, must also be considered to contribute significantly to the leucocytosis and thrombocytosis following epinephrine in some patients under these conditions. Likewise, the pulmonary circulation warrants careful study in neutropenic and thrombopenic states that are not completely explained by current theories. These data do not prove that platelets are produced in the human lung, as has been suggested by the studies of Howell and Donahue (*5*) in the dog, but merely illustrate that in some patients without panhematopenia, the lung may be stimulated to deliver platelets promptly into the circulation. The continued discrepancy between the arterial and venous platelet number suggests removal of some platelets in the peripheral circulation. Details of these studies will be published elsewhere.

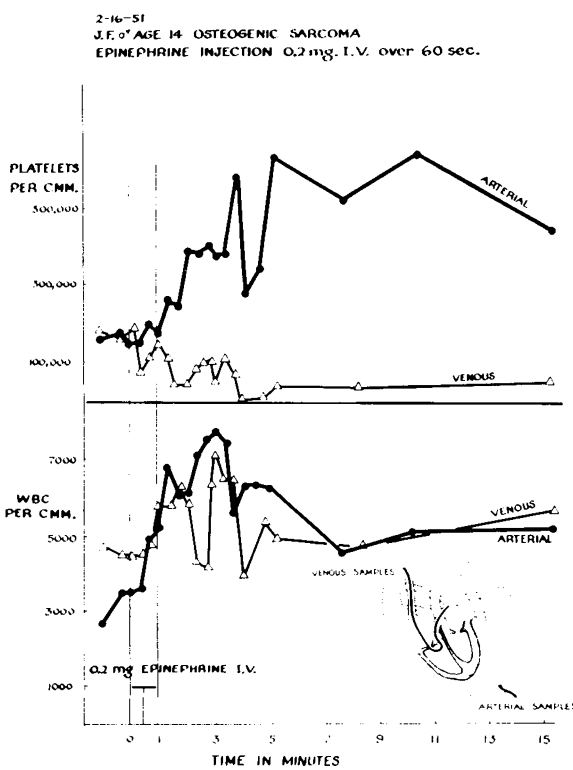

FIG. 1. Venous samples from the pulmonary conus; arterial blood from the femoral artery. There was no significant change in the red blood count in either arterial or venous blood throughout the period of study.

References

1. FREY, W., and LURY, S. *Z. ges. exptl. Med.*, 2, 50 (1914).
2. DOAN, C. A., and WRIGHT, C. S. *Blood*, 1, 10 (1946).
3. DAMESHEK, W., and MILLER, E. B. *Ibid.*, 1, 27 (1946).
4. LUCIA, S. P., LEONARD, M. E., and FALCONER, E. H. *Am. J. Med. Sci.*, **194**, 35 (1937).
5. HOWELL, W. H., and DONAHUE, D. D. *J. Exptl. Med.*, **65**, 177 (1937).

Effects of Male Hormone upon the Tail of the Slider Turtle, *Pseudemys scripta troostii*

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It is during the fourth or fifth year, usually, that the male slider turtle attains sexual maturity, as indicated by the presence of sperm in testes or vas deferens. At this time the tail grows rapidly and becomes notably longer than that of the female (*1*).

The manner in which the tail of the male is utilized in preliminary courtship (*2*) and in mating (*3*) has been described. The greater length of tail is necessary to consummate the mating process, since the length of the plastron of the male averages 13.5 cm, whereas that of the female averages 18.9 cm (based upon measurements of more than 800 specimens of each sex examined [*4*]). Measurements of the tails of skeletons of *Pseudemys* at the American Museum of Natural History also reveal that the tail of the male is definitely longer than that of the female, despite the fact that the same number of caudal vertebrae occurs in both sexes (24 ± 5).

It would thus appear that the greater length of the tail of the male slider represents a secondary sexual character and that it is subject to control by the male sex hormone. This is confirmed by the experiment to be described.

Two groups of juvenile sliders were secured for study. One averaged 5.5 cm plastron length, the other 3.5 cm. Ten of each group received pellets of testosterone propionate[1] (6.5 mg and 4.0 mg, respectively) in September 1948. Ten others of each group were retained as controls and were kept in separate aquaria. All specimens received similar care and food. Mortality averaged 20% among the larger specimens and 30% among the smaller, with no greater loss recorded among the treated turtles than the controls. The experiment was terminated in May 1950, when all animals then living were sacrificed.

[1] Generously supplied by Ciba Pharmaceutical Products.

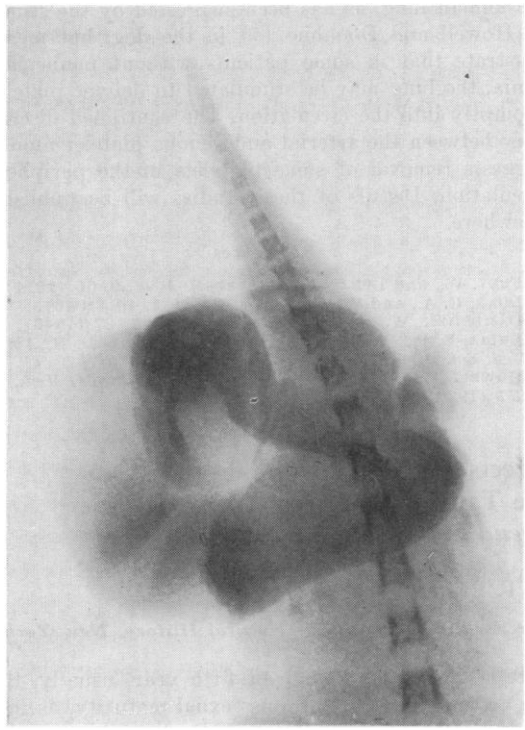

FIG. 1. Tail of juvenile turtle, treated with male hormone. × 4. X-ray photographs by Photographic Department of The American Museum of Natural History.

Despite the fact that the animals received citrus juice, cod-liver oil, and bone meal in addition to fresh beef and fish, the ossification centers of the tails of the smaller treated turtles failed to show in the x-ray photos. The soft tissues of the tails of the latter, however, responded like the larger ones to endocrine stimulation, with a comparable degree of hypertrophy.

Fig. 1 shows an x-ray photo of the tail of a turtle (plastron length, 5.5 cm) that had received 6.5 mg of testosterone propionate in September 1948 (sacrificed, May 1950). The out-bulging structure is the shadow of the penis, which was hypertrophied to such a degree that it could not be retracted into its sheath. Not all treated individuals exhibited a comparable enlargement of the penis. It was presumed that only males showed such precocious genital development. All treated specimens displayed similar tail enlargement, however. Fig. 2 portrays the x-ray shadow of the tail of a turtle (plastron length, 5.5 cm) that served as a control.

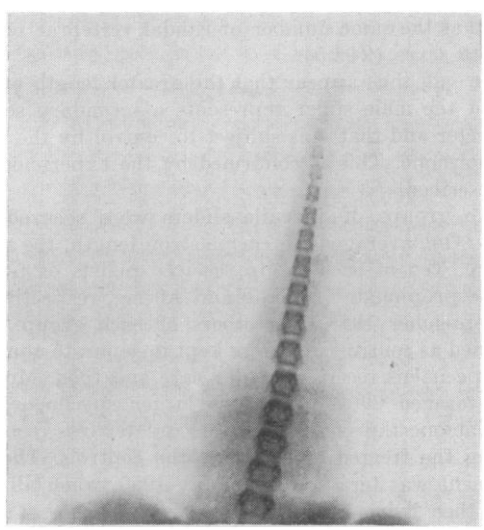

FIG. 2. Tail of untreated juvenile turtle. × 4.

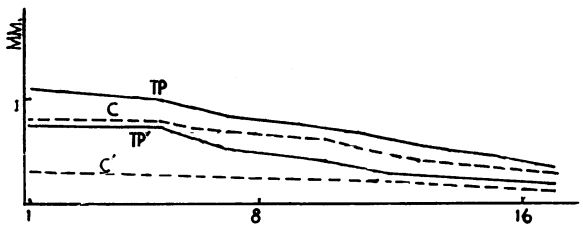

FIG. 3. ———, *TP* and *TP*¹, and — — —, *C* and *C*¹, represent the vertebrae and intervertebral spaces, respectively, of (1) juveniles treated with male hormone; and (2) those of untreated juvenile controls.

Figs. 3 and 4 compare graphically the dimensions of caudal vertebrae and intervertebral spaces in juvenile and adult sliders. In both figures the upper two graphs indicate vertebrae, the two lower (♂′, ♀′, and *C*′, *TP*′), intervertebral spaces. In Fig. 3 the structures of juvenile sliders treated with male hormone are compared with those of juvenile controls. Fig. 4 compares corresponding structures of adult male and female sliders. There is a relatively close similarity in the graphs pertaining to the adult female and the juvenile control. The same similarity holds between the adult male and the treated juvenile, but it is masked by the unequal growth of particular vertebrae, which occurs prior to the attainment of maturity.

It is possible that if older juveniles, or a longer period of endocrine stimulation, had been used a more precise duplication of the natural male hormone might have been achieved. However, there seems to be no doubt that the tissue constituents of the slider tail are markedly responsive to male hormone.

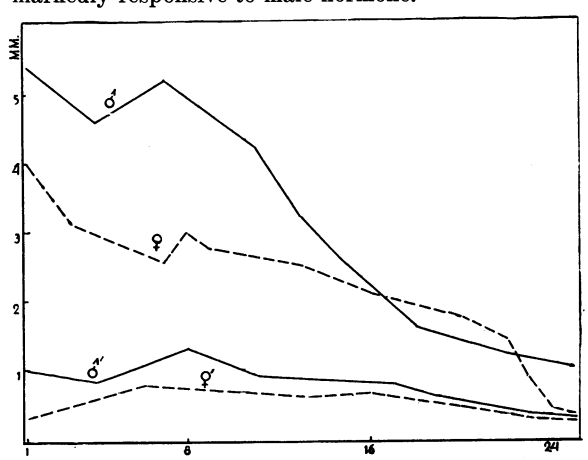

FIG. 4 ———, ♂ and ♂′, and — — —, ♀ and ♀′, represent the vertebrae and intervertebral spaces, respectively, of (1) adult males, and (2) adult females.

All the juvenile specimens that received testosterone propionate pellets also exhibited unusually long claws on the second, third, and fourth digits of the forefeet when sacrificed. This confirms a previous report (*5*) that male hormone induces hypertrophy of the second, third, and fourth foreclaws of juvenile slider turtles. Cagle (*3*), Conant (*6*), and Taylor (*7*) have indicated the manner in which the elongated foreclaws of the male slider are utilized in the preliminary phase of courtship. It has also been noted that at sexual maturity these foreclaws exceed in length those of the adult female by at least 2.5 times (*1*). It is thus evident that the middle foreclaws of the male slider, as well as the tail, represent secondary sexual characters.

References

1. CAGLE, F. R. *Copeia,* (2), 108 (1948).
2. MARCHAND, L. J. *Ibid.,* (3), 191 (1944).
3. CAGLE, F. R. *Ecol. Monographs,* **20**, 33 (1950).
4. ———. *Copeia,* (3), 149 (1944).
5. EVANS, L. T. *Anat. Record,* **94**, 64 (1946).
6. CONANT, R. *Am. Midland Naturalist,* **20**, 1 (1938).
7. TAYLOR, E. H. *Univ. Kansas Sci. Bull.,* **21**, (9), 269 (1933).

Microspectrophotometry and Cytochemical Analysis of Nucleic Acids

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The technique of microspectrophotometry, a recently developed method of cytochemical quantitative analysis whereby the spectral transmittance of a minute part of the cell nucleus is measured, can be improved from the photometrical point of view. Many workers (*1–5*), illuminate a large area of the microscopic tissue section, including the part to be measured, and select the light by a diaphragm placed at the image plane of the photomicrographic system. Owing mainly to internal reflections in the magnifying optical system, the light passing through the adjacent area of the minute part in the tissue section may cause a stray light that is added to the image of the part in question, so that the measured value of the transmittance may be enhanced. The effect, which is especially remarkable when the transmittance of the minute part is small compared with the outer illuminated area, is known in microdensitometry of photographic images of stars and spectral lines as the Schwarzschild-Villiger effect (*6,7*).

In order to reduce this effect, we have illuminated the minute part in question only, as is usually done in precision microdensitometry. Our illuminating optical system consists, as shown in Fig. 1, of a photomicrographic system that is the same as that used for magnifying, but with the reversed direction of light. An iris diaphragm of variable diameter (2–10 mm), which serves as the light source, is imaged with a reduction to 1/2000 by the system, and only a section 1–5 μ in diameter is illuminated. As ordinary microscopic condensers are not suitable for this purpose because the corrections of aberrations are insufficient, a Zeiss objective 1/12 (oil immersion) is employed. The problem of its short working distance is overcome by using a thin cover glass for the tissue section. The magnifying part of our optical system is the same as usual, with a second iris diaphragm placed at its image plane to cut out the remaining stray light, thus introducing to the photoelectric tube light from the minute part in question free from the Schwarzschild-Villiger effect. A second optical path from the light source to the phototube (Fig. 1) furnishes a check on the variation of the intensity of the light source during the measurement.

Our system has proved to be especially useful in the quantitative analysis of nucleic acids in a cell nucleus of spherical form. When the reduced image of the light source is formed at the center of a small transparent sphere, and when the dimension of the image is sufficiently small compared with the diameter of the sphere, then the length of the optical path of any beam of light passing through the sphere is the same as, and is equal to, the diameter of the sphere, being independent of the inclination angle of the beam to the optical axis, as shown in Fig. 2. Therefore, if the sphere is made up of a solution, we can determine exactly the concentration c of the solution by the formula

$$T = e^{-\varepsilon c(2r)}$$

by measuring the radius r and the transmittance T of the sphere by our system, provided that the extinction coefficient ε of the solution be known.

The following experiment has been made to exemplify the above theory. Various small spheres of 0.030 *M* aqueous solution of safranin, 2–30 μ in diameter, were prepared by mixing and agitating the solution with cedar oil; their transmittances for a monochromatic light (λ 546 mμ) have been measured, and their concentrations have been calculated, by use of the above formula. The calculated values for vari-

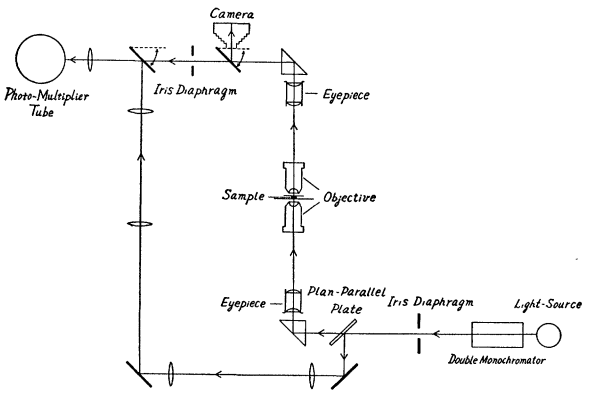

FIG. 1.

[1] The author wishes to extend his sincere thanks to Z. Koana, who has advised him regarding the Schwarzschild-Villiger effect and the method for preventing it, and who has guided him throughout the photometric work. Special indebtedness is due to A. Sibatani, of the Institute for Microbial Diseases, University of Osaka, for the preparation of the specimens, and to G. Kuwabara for help with photometric techniques.