ticularly valuable where the tumors are so resistant that administration of a massive dose of sufficient magnitude to produce complete regression is not practical. Elaborate measurements are reduced to a minimum because of the shorter period of observation required. Sampling errors arising from variation in tumor size are avoided, since such variations have little effect on the slope of the growth curve as long as the observation is confined within the interval where the growth rate remains constant. This difference is obviously due to the fact that, theoretically at least, lethal regression will not occur until all the cells in the tumor are affected lethally, whereas relative reduction in growth rate as in the present method is sufficient to indicate an effect. Frequently, resumption of growth at an accelerated rate takes place after a latent period following the administration of a sublethal dose. An example of this nature has also been observed when treatment of this tumor with guanazolo is discontinued (*3*). Such a phenomenon, when it occurs, renders both lethal regression and daily growth rate useless as criteria for the quantitative appraisal of the effect of the therapeutic agent.

As a prerequisite to the applicability of this method, both the control and irradiated tumors must grow at a constant rate for a sufficient period immediately after the irradiation. Under these circumstances, it is noted that change of slope k with dose is independent of the time interval, even though the growth of the irradiated tumor relative to that of the control tumor at any subsequent time may decrease appreciably with this interval.

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Manuscript received March 3, 1952.

The Effect of Anesthesia upon Adrenergic Blockade[1]

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In a number of instances the author has observed effects of adrenergic blocking agents in trained unanesthetized dogs which did not seem to be in accord with the pharmacodynamic effects reported for such drugs (*1*). Since the usual procedure for determining the effects of such drugs is to measure changes effected in the responses of anesthetized animals, it was deemed necessary that a comparative study be made on anesthetized and unanesthetized animals using identical techniques in both.

Epinephrine and nor-epinephrine were used as constricting agents. These were injected into the carotid artery so that only the constricting effect on blood vessels was measured. A dose of 0.1 μg/kg was used, since it causes a constriction in the blood vessels of the ear equivalent to that produced by a standard intravenous dose of 2 μg/kg. Section of the sternocleidomastoid muscle and suturing it beneath the common carotid artery make intra-arterial injection a simple procedure in trained unanesthetized dogs. Vascular volume changes in a section of the ear were measured, using a photometric technique employing a photomultiplier tube (RCA 931A) and recording the output from this tube with a string galvanometer. Mean blood pressure was recorded by a membrane manometer with its lever suspended in the light beam beside the shadow of the galvanometer string. The photomultiplier tube was activated by a white light which passed through an area of the ear measuring 5×15 mm. The light intensity was adjusted so that the control output from the tube was between 15 and 20 mv. Changes in caliber of the blood vessels in this area are recorded in arbitrary units representing 0.1-mv change in the output of the tube.

An attempt was made to select the same area of the ear for each assay. However, there were day-to-day variations in the amount of light required to produce the same activity of the phototube. This probably indicates that the volume of blood in the vessels of this area of ear varied from day to day. Moderate asphyxia produces only minimal changes in the light transmission when this technique is used and does not influence the results.

The degree of constriction produced by epinephrine and nor-epinephrine in control experiments was relatively constant. In the trained dogs after control values were established, the degree of constriction was measured after adrenergic blockade, using a β-chloroethyl amine (SY 28, 2 mg/kg) and an ergot (D.H.O. 180, 0.2 mg/kg).

The results of these procedures are shown in Table 1. Each figure represents the average of 8–10 experiments. It is quite evident that, when an animal is anesthetized, either SY 28 or D.H.O. 180 is effective in reducing the degree of constriction produced by either test compound. However, if animals are not anesthetized, adrenergic blockade has little effect on the constrictor action. The slight difference between average control responses of anesthetized and unanesthetized dogs is not significant. In this study D.H.O. 180 seems somewhat more effective in blocking constrictor action than SY 28. As little as 0.006 μg/kg of epinephrine caused a measurable constriction when injected into the carotid artery.

The results on anesthetized dogs agree with those of Folkow *et al.* (*2*), but differ from those of Bülbring and Burn (*3, 4*). I also agree with Folkow that on rare occasions one finds a dilator response following the intra-arterial injection of epinephrine. One more commonly finds dilatation in the unanesthetized dog without adrenergic blockade. Such a response may be reversed in less than ½ hr, for no apparent reason

[1] Most of the research here reported was done in the Department of Pharmacology, Temple University, aided by a grant from the Smith, Kline, & French Laboratories.

TABLE 1

VASCULAR VOLUME CHANGES IN THE DOG EAR FOLLOWING INTRA-ARTERIALLY INJECTED ADRENERGIC HORMONES

Hormone	Degree of constriction*			
	Control	After SY 28 (2 mg/kg)	After D.H.O. (0.2 mg/kg)	After both agents
With anesthesia				
Epinephrine	21.2 ± 7.1	7.3 ± 2.1	3.4 ± 2.4	0 ± 0
Nor-epinephrine	13.0 ± 2.8	2.9 ± 2.1	2.0 ± 1	1.0 ± .2
Without anesthesia				
Epinephrine	23.4 ± 6.8	24.3 ± 10.6	22.1 ± 6.8	19.3 ± 7.6
Nor-epinephrine	15.8 ± 14.2	13.7 ± 8.4	18.5 ± 8.2	15.3 ± 2.9

* Each unit represents 0.1 mv change in output of photomultiplier tube.

that the author could determine. Occasionally biphasic responses are obtained in which a slight and brief dilatation precedes the constriction.

The inability of adrenergic blockade to prevent constriction in cutaneous vessels is very striking, and this is true in spite of the fact that the blood pressure response to intravenously injected epinephrine is reversed equally in both anesthetized and unanesthetized dogs.

It seems that the normal body is able to sensitize cutaneous blood vessels to either the constrictor or dilator effects of epinephrine. This control is lost in experiments on denervated vessels and in some experiments on anesthetized animals, where it is also possible to depress the constrictor effect by adrenergic blockade. However, the unanesthetized dog apparently neutralizes the effect of the blocking drug to some extent by sensitizing the vessels to the constrictor effect of epinephrine.

Further study of the vessels in other tissues is being made in order to explain these phenomena.

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Manuscript received March 6, 1952.

Comments and Communications

Cooperation between Systematists and Experimental Biologists

IN THE recent excellent collection of papers making up the Michaelis memorial volume, *Modern Trends in Physiology and Biochemistry* (New York: Academic Press [1952]), produced by the staff of the physiology course at the Marine Biological Laboratory, Woods Hole, there appears a footnote (p. 339) by Dr. Wald which poses a problem and a challenge to those who would like to see a healthy cooperation between experimental biologists and their fellow-workers in taxonomic fields. This footnote, although extremely humorous and to the point, reflects a widespread, although by no means universal, state of mind among experimental biologists, and, indeed, complaints of this sort have of late become as familiar around Woods Hole as the cries of the sea gulls, but not so easily ignored. The gist of the difficulty seems to be that repeated changes in the names of animals long used in experimental work have caused so much confusion that busy physiologists simply can no longer follow them and might as well ignore them. The examples cited of the mandrill and Guinea baboon, and of *Limulus* versus *Xiphosura,* hardly represent contributions by taxonomists to a stable nomenclature, but to conclude from such extreme cases that name changes in general must be deplored would seem to indicate that physiologists are not fully aware of the problems of the systematist, nor of the conventions of zoological nomenclature. It is equally true that on numerous occasions systematists have revised the names of animals in very common experimental (or commercial) use without publishing clearly in journals accessible to experimentalists the reasons for the changes.

The problem expressed by Dr. Wald affects experimentalists and taxonomists alike, and at some risk of being caught in the ensuing cross fire, I shall try to point out certain reasons for the present lack of cooperation, and to suggest a positive step toward a lessening of the existing confusion. Not being a taxonomist, I should make clear that I am interested, not in the oversimplification of genuine nomenclatural problems, but rather in promoting a workable and beneficial relationship between experimentalists and taxonomists.

Experimental biologists should realize that there are two very different aspects of the problem of naming organisms. One is the matter of *nomenclature,* which is at its simplest the task of assigning a name to each distinct species of plant or animal.