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23 July 1973

Pavlou and Clayton appear to have missed the point of our discussion of pollution in coastal waters. In our figure 11, we used data from McClure and Barrett (*1*) as an illustration of the fact that "wind- and wave-induced surface currents tend to produce circulation patterns that favor the retention of particulate material near the coast . . . , whereas biological scavenging and absorption by suspended particles (both biogenous and inorganic) concentrate dissolved pollutants in coastal waters" (*2*). In this context the figure we used serves as a valid illustration. We had no intention of entering into a discussion of methods of normalization of biological data, a procedure that is subject to considerable controversy. However, even when the data in figure 11 (*2*) are normalized, as suggested by Pavlou, "the gradients are quite definitely still there! The 'hot spot' still shows apparent concentrations fivefold higher than the average over the whole grid" (*3*).

Pavlou and Clayton comment that a statement in our discussion of mixing is redundant. The concentration gradient can only be described as a "function of the mixing process" when both the mechanics of mixing and the sources or inputs are processes which do not vary independently. For example, a concentration gradient established at some time at a fixed point by mixing processes may change at some later time if subjected to different processes or to variations in their intensities. Further, a concentration gradient previously established by mixing processes at some point may change after advection to some other point where there are different sources and different mixing processes. Also, the source is not always in the form of point sources, as in the case of river runoff or ocean outfall; occasionally, offshore winds can introduce concentrations of pollutants over large water areas. If these contaminants remain in the surface waters, they will tend to be contained against the coast by the prevailing northwesterly winds. Thus, our statement is not redundant.

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19 March 1974

Conditioning or Control?

Harris *et al.* (*1*) described an "instrumental" conditioning procedure in which reinforcement (food-reward and shock-avoidance) was contingent upon specified elevations of the diastolic blood pressure of baboon subjects. The observed significant elevations for each of the four subjects were of large magnitude (30 to 40 mm-Hg) and were sustained over 8 to 10 weeks. The authors stated that the response change was "directly and specifically" a result of the programmed contingencies of reinforcement. In my view the data do not unequivocally support this conclusion.

Demonstrative conditioning of autonomic nervous system activity in general, and cardiovascular responses specifically, requires that the autonomic response of interest be neither an unconditioned response nor mediated by somatic activity (that is, skeletal muscle activity or respiration) (*2*). The possibility of such mediation has been shown for human and infrahuman subjects (*3*). A variety of control tactics has been specified for minimizing such confounding effects, include: bidirectional control, paralysis of skeletal musculature by curariform drugs, yoked-control subjects, and the differential conditioning of a presumed mediating response (*4*). Harris *et al.* report that two additional animals were exposed to identical reinforcement contingencies for *decreases* in diastolic blood pressure, thus completing the bidirectional procedure. If bidirectionality was not observed, then unconditioned mediators cannot be dismissed. The authors reported that this procedure failed to produce decrements after 6 months of training.

Animals reinforced for increments in diastolic blood pressure received a mean of two electric shocks and 25 food pellets per hour. Similar data for the control group were not presented, and it is likely that these animals (not meeting the response requirement) received considerably more shocks and less food. Such an inequality might provide the basis for unconditioned or classically conditioned responses mediating the blood pressure response and entirely account for the differences in blood pressure between the group reinforced for increments and the group reinforced for decrements. Additionally, although short-term peripheral mediation might be ruled out, failure to include concomitant measures of respiration and skeletal muscle activity does not allow specification of possible long-term mechanisms mediating the increments in diastolic blood pressure.

In the absence of such control procedures, it appears judicious to adhere to the recognized distinction proposed by Black (*5*) between *control* and *conditioning* of autonomic responses, the latter reserved for response changes directly attributable to the response-reinforcer contingency. Thus the effects obtained by Harris *et al.* are accurately described as representing control, not conditioning.

This criticism does not deny the utility of the experimental model proposed by Harris *et al.*, but rather urges an important distinction in the study of behavioral-physiological mechanisms that mediate cardiovascular activity.

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28 January 1974

The conditions under which the term "conditioning" is used continue to be of concern to behavioral scientists, and now, with the advent of "operant autonomic conditioning," the specification of these conditions is further complicated. For some, the elimination of certain somatic "mediators" (for example, skeletal muscle activity and respiration) will suffice. For others, more stringent requirements (for example, the elimination of "cognitive states," or central nervous system activity related to skeletal muscle activity) are placed upon the use of the term "conditioning." The preparation of a subject to satisfy conditions of "pure" autonomic conditioning remains unspecified, unattainable, and probably unnecessary.

What precisely is a "mediator" and how many of "them" are there that must be controlled? How much of the organism is to be left intact and functioning? Is the central nervous system to be left undisturbed? All this is not to say, however, that variables such as respiratory, somatic, and central nervous system activity are unimportant or should not be systematically studied. Both behavioral and physiological mechanisms involved in the conditioning of autonomic responses invite creative research. Whether or not contingencies placed upon respiration or some aspect of skeletal muscle activity will produce the same magnitude change in blood pressure over the same time course as did the contingency placed upon blood pressure, remains an empirical question.

A second point raised by Millard pertains to the unconditioned effects of food and shock stimuli upon blood pressure. The more common concern in this regard is that such stimulation (particularly from the electric shocks) is directly responsible for the observed blood pressure rise in these animals. Since the animals exposed to the blood pressure lowering contingency also received food, shock, lights, and so forth, but evinced no sustained blood pressure elevation, we feel that this indicates that the blood pressure increases in the other animals were not attributable to the unconditioned effects of these stimuli. Further, it seems unlikely that a situation involving somewhat more shocks and less food (the situation for the blood pressure lowering animals) would be associated (that is, as an unconditioned effect) with a lower pressure, and a situation involving fewer shocks and more food (the situation for the blood pressure raising animals) associated with higher pressure. The contingencies aside, it would seem that the former situation would be more "stressful" than the latter.

Lastly, there is a special irony associated with the distinction offered between the terms "control" and "conditioning." For many years, and even now, operant conditioners have used the two interchangeably with phrases such as "behavioral control," "schedule control," or "stimulus control of behavior," usually reflecting the operations of various conditioning procedures. Our use of the term "instrumental conditioning" ("operant" would have been better) referred simply to a set of procedures that we have employed many times with respect to several response dimensions, including rate, intensity, and duration, for animals bar pressing, key pecking, lever pulling, or vocalizing. Since special preparation of the subject (that is, curarization or respiratory regulation) was never necessary before in order to use the term "conditioning," we extended the same usage when working with the blood pressure response. This, of course, raises additional questions regarding the definition of a "response"; and a reexamination of the use of all these terms—"conditioning," "control," and "response"—would undoubtedly be instructive. While we would find no problem substituting the term "control" for "conditioning," I'm afraid it's because we see the similarity rather than the "important distinction" between the two.

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4 March 1974