

of living brains. The parallels between animate and inanimate thinking machines are clearly shown, but they are not forced.

Berkeley is writing about revolution, but Pfeiffer is writing about evolution: "The evolution of all other living things has depended on changes in their bodies. But man could evolve indefinitely without any such changes, with the brain he has now. Our kind of evolution depends on cultural changes, on what we learn, on the things we build. In a basic sense, human evolution is the evolution of machines, and of these, computers are the most significant. Perhaps more than anything else, the design of artificial-intelligence systems will determine our future as a species."

These books have much to recommend them to both the scientific specialist and the nonscientific layman.

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## Cognitive Dissonance

**Deterrents and Reinforcement.** The psychology of insufficient reward. Douglas H. Lawrence and Leon Festinger. Stanford University Press, Stanford, Calif., 1962. vi + 180 pp. \$4.75.

This book provides the converse of the familiar and much-debated attempt to apply theories based on animal research to the behavior of humans; it applies the theory of cognitive dissonance, which was originally developed for human attitudes and opinions, to several aspects of the behavior of rats. It deals principally with resistance to experimental extinction, taking as its point of departure the assertion that no previous theory has satisfactorily accounted for the fact that partial reward, delayed reward, and high effort during learning produce responses that are extremely persistent in the face of repeated nonreinforcement.

Illustratively and sketchily, the assumption is this: a rat that is rewarded on only some of its trial runs down an alley to a goalbox will experience cognitive dissonance on those trials when it is not rewarded because the facts, running-for-food and not-getting-food, are in a dissonant relationship to each other. The authors assume that the rat is motivated to reduce this dissonance and that it will do so, as best it can,

by finding extra attractions in the goalbox or in the activity itself. Then, if the original reward is removed, these extra attractions will provide a form of secondary reinforcement to maintain the response.

The book explains this approach in various situations and reports the results of a series of 15 studies designed to evaluate it. By and large the results of these studies are consistent with the stated expectations.

Beneath the surface there are several difficulties, some of them serious. For example, it is not clear whether dissonance results from rewards that is insufficient to the work done or to the level of reward expectation. In any event, expectations play an important role, but their properties are not described. Furthermore, it is not always clear when extra attractions will develop; for example, rats rewarded on every trial during learning could find extra attractions in the goalbox as a result of dissonance encountered on early extinction trials. Such uncertainties make it impossible to draw rigorous conclusions.

However, the authors are aware of many of these problems, and they are reasonably modest in their claims. A tentative and incomplete theory may be very stimulating to researchers. This will almost certainly be true of dissonance theory applied to animal behavior.

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## On Ways To Study Man

**Methodology in Human Genetics.** W. J. Burdette, Ed. Holden-Day, San Francisco, Calif., 1962. 436 pp. \$4.

This is a collection of 15 papers presented at the symposia held in Salt Lake City, 13 and 14 May 1960. The date of the meeting is not mentioned in the preface of the volume; consequently, the reader may not realize that two full years elapsed between presentation of the papers and their appearance in print. Such publication lags must be remedied in the future.

One common characteristic of these papers is, as the title implies, emphasis on methodology rather than on factual findings. Space here permits only bare mention of the author's name and one or two key words from

the title of his paper. Thus, Lilienfeld: sampling and tests; Morton, segregation and linkage; Crow: selection; Steinberg: special cases; Newcombe: linked records; Novitski: computers; Roberts: inherited diseases; Herndon: empiric risks; Cox and MacLeod: susceptibility to infection; Neel: mutations; Ford: cytogenetics; Puck: cell cultures; Sutton: metabolic defects; Hill: hemoglobin structure; Boyd: soluble antigens. Motulsky, Gowen, Tjio, Patau, and others, made substantial contributions to the discussion.

Of course, methodology, the unifying theme of the symposium, has no concrete meaning by itself. Hence, the methodology presented in this volume covers mathematical formulations, sampling procedures, systems of filing records, statistical grinding, clinical diagnosis, microtechniques, tissue culture directions, and biochemical analyses. The papers are no more and no less heterogeneous than those in many other symposium volumes. Most of the papers are very general reviews, but some are carefully organized; some are hastily written, and a few others could have been given a few years earlier without any substantial revision. Nevertheless, I think each review serves a useful purpose.

Reading Novitski's account of computer programming is like reading a story on jaguar hunting in Paraguay. Even those who have never hunted, do not know where Paraguay is, and cannot be sure whether jaguar is a big cat or a motor vehicle, will find the story interesting and understandable.

Earlier genetic studies on susceptibility to infectious diseases in man have not been as fruitful as those on plants and animals where strictly controlled experimentation is possible. The impression that infectious diseases are not amiable to genetic analysis is partially due to the fact that the resultant disease itself has been taken as the object of study. Cox and MacLeod, however, have collected a host of examples to show that, if we study the underlying conditions of a particular organ (presumably having a genetic basis), which lead eventually to higher incidence of infection, then the phenomenon is just as amiable to genetic analysis as any other conditions. Outstanding examples are: In the case hypogammaglobulinemic children, the susceptibility to infections is due to a defective immune mechanism that is genetically controlled; among children with cystic fibrosis of the pancreas the suscep-