Galaxy M 31

In his report "Radio map of the Andromeda galaxy" (24 July, p. 389), John M. MacLeod reported the results of observing the nearby galaxy M 31 with the new University of Illinois radio telescope. At about the same time a paper of mine appeared in the *Astrophysical Journal* (**139**, p. 1045) analyzing the spiral structure of M 31. Putting the results of the two investigations together now reveals an unexpected and intriguing puzzle.

My new star arm and schematic spiral pass inside the old spiral arm point N 4. As a result the three maxima of 610 mcy/sec radiation coincide with three major axis crossings of spiral arms (N 2, S 3, and N 4). But this is one single arm that is outlined. The symmetrically opposite spiral arm does not seem to register in the mapping. An even more startling point is that the arm which MacLeod observes to radiate is the arm which I suggested was attached to M 31's dwarf elliptical companion, M 32 (at S 5). If my estimate of the situation is correct, then we have a problem which involves the nature of galaxies and unsuspected physical phenomena.

HALTON C. ARP
Mount Wilson and Palomar Observatories, Pasadena, California

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Learning and Performance: A Distinction

Woolley and van der Hoeven have recently published two reports (*1*, *2*) which purport to show changes in "learning ability" as a function of the amount of serotonin in the brain. In most learning experiments the dependent variable (change in behavior) and the independent variable (in the case of the Woolley and van der Hoeven study, changes in brain serotonin) are directly observable, whereas the intervening variable (learning), which links the independent with the dependent variable, cannot be observed directly. Learning is usually reflected by changes in performance, but it is obvious that changes in performance can arise from sources other than the learning process (such as fatigue).

In the initial Woolley and van der Hoeven study experimental animals were treated with compounds which manipulate the serotonin content of the brain. After these manipulations, the animals, along with untreated controls, were permitted to explore a T maze for 2 minutes and were then immediately given ten test trials in the same maze. Woolley and van der Hoeven attributed differences between the experimental animals and the controls to changes in "learning ability" induced by changes in the serotonin content of the brain (*1*). The choice of the term "changes in learning ability" is not justified by the experimental design, since the drug effect was not restricted to the learning period. In fact, they say later in the same report that there were no differences in performance when the mice were retested "several days after cessation of the treatments." The absence of differences between groups after the drug effects were dissipated would seem to indicate that the treated mice showed neither better nor poorer "learning ability" than the untreated mice and that it was performance, rather than learning, that was affected by the experimental conditions.

Although Woolley and van der Hoeven acknowledged in the first report that the compound they employed to *decrease* cerebral serotonin decreased catechol amines also, in referring to this experiment in the second report they say that they related the increases in learning ability "specifically to the deficiency of serotonin." It would have been well to attempt to decrease norepinephrine selectively with a compound such as α-methyl-*m*-tyrosine (*3*).

DONALD E. MCMILLAN
Department of Psychology, University of Pittsburgh, Pittsburgh 13, Pennsylvania

References

1. D. W. Woolley and Th. van der Hoeven, *Science* **139**, 610 (1963).
2. ———, *ibid.* **144**, 883 (1964).
3. B. B. Brodie and E. Costa, *Psychopharmacol. Serv. Center Bull. 2* (1962), pp. 1–25.

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We were well aware that some of the means we used to alter the serotonin content of the brains of the animals also alter the catechol amine contents, and mentioned this fact in our papers. We were also at some pains to determine whether the observed changes in learning ability were due to the changes in serotonin or to the concomitant changes in catechol amines. To do this we used independent methods of changing the hormone contents. Catechol amines were caused to increase in the brains by administration of dihydroxyphenylalanine. This did not change the learning ability, whereas by contrast, when serotonin was caused to increase specifically by administration of hydroxytryptophan, the change in learning ability was found. We did not use α-methyl-*m*-tyrosine because there are conflicting statements in the literature as to whether it causes changes in catechol amines alone, or in serotonin as well as in catechol amines.

McMillan also criticizes us for not distinguishing between learning and performance. The first of our two papers mentioned our concern about this question and told something of what we did about it experimentally. The testing procedure was designed so as to give some information about this point. For example, by changing the time allowed for learning, one can get information about whether the change in performance is the result of an effect on learning or whether it is due to other causes. The fact that the animals did not remember for a long time what they had learned did not mean they had not learned it. There is a short-term memory and a long-term memory. We were studying a short-term memory. Because a report in *Science* must be limited in length, it was not possible to say much about these questions or about other important points.

We do not want to give the impression that we feel that either of the very complicated problems raised by McMillan has been settled completely. We wish only to point out that we were very much aware of them in our work, and did something to throw light on them.

D. W. WOOLLEY
T. VAN DER HOEVEN
Rockefeller Institute, New York 21

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