or inhibitory portions of their receptive fields.

The original McCollough concept of an edge detector, although somewhat more complex than a dipole, was likewise presented as a straight-line model. It assumes that direction of orientation is the main property of an edge (*1*). To add direction of curvature as another property to be analyzed by edge detectors would be to raise formidable questions about the explanatory value of this construct.

A particularly attractive hypothesis, that of channels specialized for the detection of the spatial frequency of lines in a grating (*5*), can easily encompass many of the results obtained by the McCollough procedure. Yet it, too, is a model based on rectilinear arrays that are tuned for direction of orientation. Inasmuch as the curvature-dependent aftereffects reported here cannot be attributed to orientation, that model also fails to provide any explanation for my results.

Where does this leave us? Neurophysiologists are finding that the processing of visual information takes place at all levels within the visual pathways (*9*). Such features as color, orientation, spatial frequency, contrast, depth, and motion are selectively effective as stimuli for specialized neural detectors. Experiments based on the McCollough effect, although restricted to the case of straight-line arrays, have already demonstrated response contingencies between color and tilt or orientation (*10*), spatial frequency (*8*), and direction of motion (*11*) of the lines. The results reported here point the way toward more general feature analysis. In particular they may stimulate electrophysiologists to look for cortical cells for detecting the degree and direction of curvature (*12*).

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12. D. H. Hubel and T. N. Wiesel [*J. Neurophysiol.* **28**, 229 (1965)] have described hypercomplex cells in the visual cortex of the cat that are of particular interest here. Some of these cells respond preferentially to angles and curves. As the authors put it, these cells "can, in a sense, serve to measure curvature; the smaller the activating part of the field, the smaller the optimal radius of curvature would be." Similar cells were also found in the monkey (*6*). Such cells, if they were also selectively responsive to colors, could well mediate the effects described in this report. K. D. White reports that "aftercolors from inspecting chevron patterns obey similar principles to the aftercolors from inspecting patterns of curved lines" (program of the annual meeting of the Association for Research in Vision and Ophthalmology, Sarasota, Fla., May 1973, p. 35).
13. These experiments were conducted in major part at the Physiological Laboratory of the University of Cambridge while the author was a Guggenheim Fellow on sabbatical leave from Brown University. I think Dr. Fergus W. Campbell for laboratory facilities and C. Hood and P. Starling for technical assistance.

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Hurricane Seeding Analysis

In the article "The decision to seed hurricanes" by Howard *et al.* (*1*) it is stated in the subtitle that "On the basis of present information, the probability of severe damage is less if a hurricane is seeded." In my opinion present knowledge does not support such a statement because the results of studies of this problem do not provide a unique answer. Consequently, no conclusions can presently be made about the economic effects resulting from seeding hurricanes.

The data available from seeding experiments (such as those from Hurricane Debbie and possibly Hurricane Ginger) are too few for a statistical analysis to yield confident conclusions. Furthermore, the results of the numerical model studies referred to by Howard *et al.* conflict with results which I reported (*2*). In fact, if the method of Howard *et al.* is applied to my results, the conclusion reached is the opposite of that reached by Howard *et al.*, as I show below.

The standard deviations adopted here are the same as those in Howard *et al.* for all three hypotheses concerning the effect of seeding (H_1, reduction of the maximum wind; H_2, no effect; H_3, increase of the maximum wind). The probability distribution for the wind speed if the hurricane is seeded, w', if H_2 is true, is the same as that of Howard *et al.* (*3*):

$$P'(w'|H_2) = P(w'|H_2) = P(w) = f_N(100\%, 15.6\%) \quad (1)$$

where w is the wind speed of the unseeded hurricane (*4*).

Using the results of the numerical experiments presented in (*2*) I assign the following probability distribution to w' for the case that H_3 is true

$$P'(w'|H_3) = f_N'(107\%, 18.6\%) \quad (2)$$

The probability distribution employed for w', if it is considered that H_1 is true, is

$$P'(w'|H_1) = f_N'(95\%, 18.6\%) \quad (3)$$

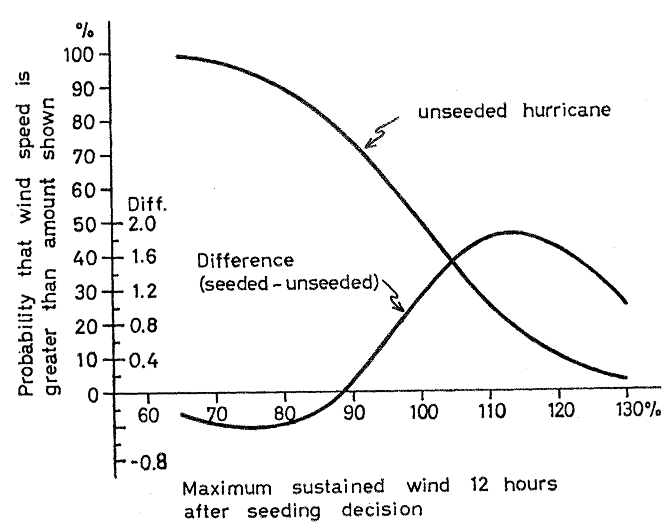

Fig. 1. Probability distribution on 12-hour wind changes for the unseeded hurricane, and the difference in probability distributions between the seeded and the unseeded hurricane.

Therefore the probability density function for the Debbie results, if hypothesis H_i is true, now becomes

$$P'(69\%, 85\%|H_i) = \begin{cases} 1.4996 & i=1 \\ 0.5716 & i=2 \\ 0.2827 & i=3 \end{cases} \quad (4)$$

Now, considering the deductions made in (*2*)—on the basis of physical reasoning and the results of numerical model experiments which definitely indicate an effect of intensification by seeding—I assign the pre-Debbie probabilities

$$\begin{aligned} P'(H_1) &= .0227 \\ P'(H_2) &= .7500 \\ P'(H_3) &= .2273 \end{aligned} \quad (5)$$

whereas in Howard *et al.* the corresponding set is

$$\begin{aligned} P(H_1) &= .15 \\ P(H_2) &= .75 \\ P(H_3) &= .10 \end{aligned}$$

Hence, the pre-Debbie odds that seeding has no effect are the same in set 5 as in Howard *et al.* However, $P'(H_3)$ is taken to be one order of magnitude larger than $P'(H_1)$ to reflect that, if seeding affects the intensity at all, an increase of the maximum wind is expected.

When sets 4 and 5 are introduced in Bayes' equation the posterior probabilities become

$$\begin{aligned} P'(H_1) &= .0647 \\ P'(H_2) &= .8131 \\ P'(H_3) &= .1222 \end{aligned} \quad (6)$$

whereas in Howard *et al.*

$$\begin{aligned} P(H_1) &= .49 \\ P(H_2) &= .49 \\ P(H_3) &= .02 \end{aligned}$$

Set 6 implies that, since the Debbie results, the odds are about 4 to 1 that seeding has no effect, and if seeding does have an effect the odds are 2 to 1 for wind intensification rather than wind reduction.

Finally, I can compute the probability distribution on wind speed [from Eqs. 1, 2, 3, and 6 above and equation 4 in (*1*)]. The difference in probability between the seeding and not-seeding alternatives is so small that it is hard to show it in a plot of the complementary cumulative distribution functions of those two alternatives. Instead, I plot this function for the not-seeding alternative and the difference (the function for seeding minus the function for not-seeding) in Fig. 1. I find that the probability for intensification (wind speed more than 100 percent of the initial wind speed) if a hurricane is seeded is .511; if the hurricane is not seeded the probability is .500 [in (*1*) these values are .36 and .50, respectively]. The probability of intensification by 10 percent or more is .278 if a hurricane is seeded and .261 if it is not seeded [.18 and .26, respectively, in (*1*)].

Furthermore, for any particular wind speed larger than 88 percent of its initial value, the probability that this speed will be exceeded is greater if the hurricane is seeded than if it is not seeded. For wind speeds less than 88 percent of their initial values the situation is reversed; however, the difference in this interval is much smaller in magnitude than it is in the former interval.

Since the analysis given above may be considered to be as soundly based as that in (*1*), it shows that the available data are too sparse to yield a statistical basis for conclusive statements. I suggest that the method of statistical analysis (possibly somewhat modified) should be used to investigate the requirements on reliability and volume of results from model studies and field experiments in order to permit confident conclusions and recommendations.

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3. The same notation is used here as in (*1*). For convenience, some of the data given in (*1*) are repeated; the probabilities from that article are designated by *P* and those of the treatment given here by *P'*.
4. As in Howard *et al.* [reference *13* in (*1*)] a probability distribution on a quantity x is denoted $P(x)$, and a probability distribution of the normal or Gaussian family specified by its mean m and standard deviation σ is denoted $f_N(m, \sigma)$.

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In the concluding section of our article we stated: "The results of a decision analysis depend on the information available at the time it is performed. Decision analysis should not be used to arrive at a static recommendation to be verified by further research, rather it should be used as a dynamic tool for making necessary decisions at any time." We are pleased that Sundqvist finds our analysis a useful format in which to present his views regarding the results of hurricane modification. He has succinctly summarized his opinion in the form of a prior probability distribution and then used the Debbie experimental results to develop consequent probability distributions for the wind speed, both with and without seeding. His pre-Debbie probability assignment was that there was a 75 percent chance of no seeding effect, and that if there were an effect, the odds were 10 to 1 that it would be deleterious. The Debbie experiment is not sufficient to overcome this pessimistic prior probability distribution: a decision-maker who subscribed to Sundqvist's view would not wish to attempt operational hurricane seeding at this time.

Our analysis was based on the best information we could obtain from U.S. hurricane modification experts. As decision analysts we cannot comment on Sundqvist's differing opinion, except to say that our information sources were aware of his work and did not subscribe to his views. Further dialogue between Sundqvist and the community of U.S. hurricane modification experts would be appropriate to determine whether the latter see any new reason to modify their judgments.

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Stable Limit Cycles in Prey-Predator Populations

In a recent report (*1*) May discussed several mathematical models of prey-predator population interactions, all variants of the Kolmogorov model (*2*). May attributed to Kolmogorov the statement that this model possesses either a stable equilibrium point or a stable limit cycle (*3*). Kolmogorov (*2*) has remarked, however, that under his own hypotheses there may be several possible configurations, one of which is an unstable equilibrium point that is surrounded by an uncountable number of neutrally stable periodic solutions (hence neither a stable equilibrium point nor a stable limit cycle).

In the same report (*1*), May claimed that his interpretation of Kolmogorov's results holds under even more general