measurement to measurement during the same night; (3) if the polarization was constant in amount and angle, then measurements of $p(\theta)$ at various θ should fit on a $\cos 2(\theta-\theta_0)$ curve. In fact the least-squares cosine fit is poorer than the probable errors of each individual measurement would lead one to expect (see the last column of Table 1). Taken together, these observations strongly suggest that the linear polarization of the main pulse is variable over a time scale of the measurement time (minutes).

In a similar manner the linear polarization of the secondary pulse was measured, and in the same way variability is suggested, though not so strongly. There is no very evident correlation between the polarization of the two pulses.

We attempted to measure the polarization within different phases of the main pulse. Because of the saturation in the i.f. converter already referred to, the results obtained (see bottom of Table 1) can only be very tentative. As far as they go, they do confirm a clockwise rotation of the polarization vector as one moves from beginning to end of the pulse as suggested by Warner *et al.*[8].

Circular Polarization

Observations of circular polarization of the primary pulse were made on February 21 (diaphragm 6·8 arc s) and March 12 (8·1 arc s). A 5·08 × 5·08 cm plastic quarter-wave plate was used (Bausch and Lomb No. 31–52–62–40). The quarter-wave plate was rotated over 90° between observations, to have one of the axes in either one of two position angles, $\theta=121°$ or 211°, in a mounting block immediately preceding the Wollaston prism, which was kept in the $\theta=166°$ position. It is essential that the prism be at 45° to the fast and slow axes of the quarter-wave plate, while the actual position angle of the quarter-wave plate plus prism combination is of no importance. Let the integrated area under the pulse, not including the background, of the first channel at $\theta=121°$ be denoted by $i_1{}^a$ and that of the second channel $i_2{}^a$; at $\theta=211°$ the areas are $i_1{}^b$ and $i_2{}^b$. If $A=i_1{}^a/i_2{}^a$ and $B=i_1{}^b/i_2{}^b$, it can be shown that the percentage of the incident light that is circularly polarized is $P_0=100\ (\sqrt{A}-\sqrt{B})/(\sqrt{A}+\sqrt{B})$. This method of reduction allows for different channel sensitivities and apparent brightness variations.

Table 2. CIRCULAR POLARIZATION OBSERVATIONS (PER CENT)

Feb. 21, 1969	Mar. 13, 1969
+0·5	−13·8
−5·7	−2·7
+9·8	−5·7
+0·6	−1·0
	+2·2
	−6·0

Table 2 gives the measurements of circular polarization. The straight average of computations gives $P_0=2\pm1$ per cent (p.e.). Separate reductions for leading and trailing halves gave no different result. The background was also found to have a negligible amount of circular polarization.

The lack of circular polarization is of great interest with respect to the theory of Chiu, Canuto and Fassio-Canuto[10], which predicts complete circular polarization in the optical band. The recent discovery of X-ray pulses[11] would also seem to invalidate the application of their theory to *NP* 0532, because they predict only two emission bands, including the radio.

The apparently rapid variability of linear polarization makes interpretation difficult. These measurements need to be repeated with a telescope of the largest aperture and focal length, in order to shorten the time of the measurements and reduce the nebular background noise.

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Moiré Effect from Random Dots

by

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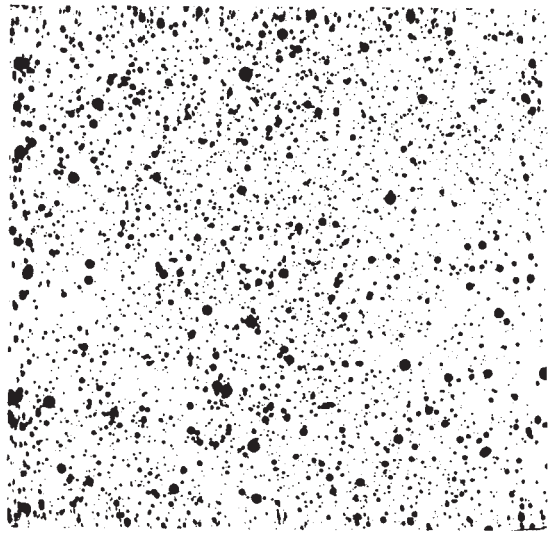
The appearance of circular Moiré fringes when a random dot pattern is superimposed on itself provides new evidence that the human visual process may include the computation of local autocorrelations by excitation of line detectors.

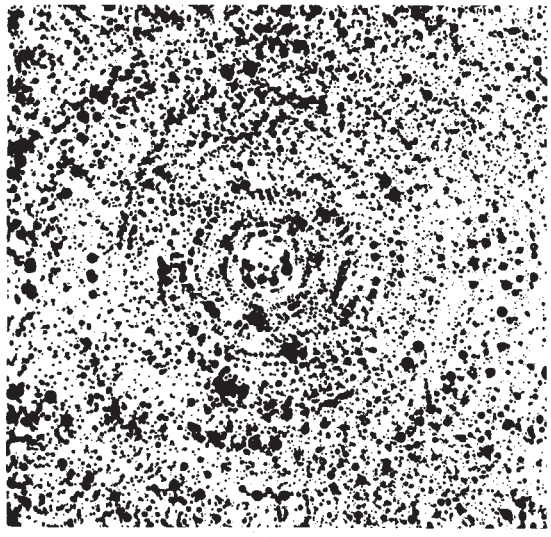
If a random dot pattern (Fig. 1*a*) is superimposed on itself, rotated a small amount and viewed monocularly, concentric circles about the point of rotation are immediately perceived (Fig. 1*b*). If this rotation is gradually increased, the circles begin to disappear, the outermost ones first, and an unstructured pattern of twice the initial density is eventually observed (Fig. 1*c*).

This effect demonstrates the ability of the human visual system to detect local correlations in the presence of noise, and to combine local correlations from different regions of the visual field in such a manner that a simple percept is formed. Below, we consider briefly possible visual information processing schemes which may achieve this result. We first consider a biological model and then a more general mathematical representation of the system.

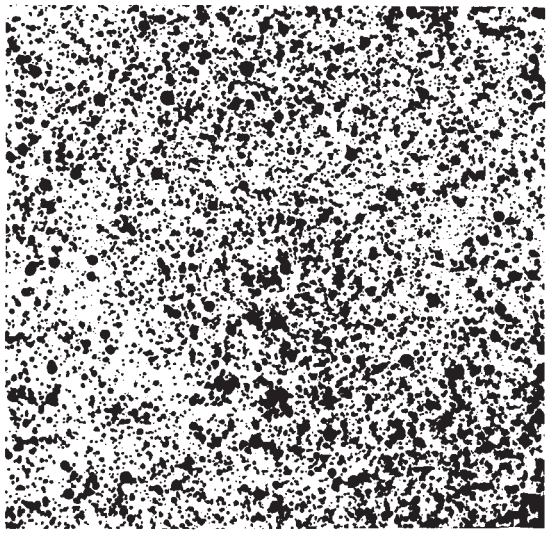
It is possible that the first step in the construction of a circular percept is the excitation of line detectors in the visual cortex[1,2]. For each dot, D, in Figs. 1*b* and 1*c*, there is a corresponding dot, D', which lies along the circumference of a circle centred at the point of rotation. If D and D' fall in the excitatory field of the same line detector and if, in addition, the number of dots falling in the inhibitory field of this line detector is sufficiently small, we would expect excitation of this line detector to occur. In the neighbourhood of D there are other randomly distributed dots and we would anticipate that some other cortical detectors, specific for lines lying in randomly distributed directions, would also be excited. If, however, we consider the area surrounding D, and if the rotation is not too great (as it is in Fig. 1*c*, for example), we would expect



a



b



c

Fig. 1. *a*, A random dot pattern, constructed by spraying black paint from an aerosol can on white paper. *b*, The pattern resulting when a transparency of *a* (made with a 3M Thermo-Fax) is superimposed on this figure and rotated 6·5°. *c*, The pattern resulting when a transparency of *a* is superimposed on it and rotated 24°.

to find that many more line detectors which lie along the circle centred at the point of rotation would be excited than line detectors which lie in any other direction. In order to perceive circles the visual system must now detect the line segments falling along the circumference of the circles centred at the point of rotation, and "disregard" the line segments lying in random directions.

Some recent studies of the visual system of cats done by Hubel and Wiesel suggest a way in which this may be accomplished[2,3]. This work indicates that cells which are excited by lines having the same orientation but which have somewhat different neighbouring and overlapping retinal fields are located in the same column in the visual cortex. When viewing Fig. 1*b* we would therefore expect many detectors to be excited in the column which is specific for lines which lie in the area around D and along the circumference of the circle centred at the point of rotation, whereas the other columns which are specific for lines lying in the area about D, but in other directions, would have proportionately fewer detectors excited. If we now assume that each column in the visual cortex functions as a summation device with a threshold (in particular, note the discussion of the functions of the complex cells in refs. 2 and 3), we may detect the signals from correlated dots and suppress the detection of signals from noncorrelated ones. As support for this hypothesis, we note that if we limit the possible summation area by exposing only a small area of Fig. 1*b*, we can no longer detect the correlations present.

The preceding discussion is necessarily speculative because of the incomplete evidence concerning the functions and structure of the visual cortex. The detection scheme which was outlined may, however, be readily put into a more general mathematical form. We denote the position of the ith dot by the vector $\mathbf{R}_i$, drawn from the point of rotation in Figs. 1*b* and 1*c* to the centre of the ith dot, and define $G_i(\mathbf{R})$ to be the local autocorrelation function about $\mathbf{R}_i$, that is the probability that there is another dot at $\mathbf{R}_i+\mathbf{R}$. That is to say,

$$G_i(\mathbf{R}) = \sum_{j \neq i, |R_j - R_i| < a} \delta(\mathbf{R} - \mathbf{R}_j + \mathbf{R}_i)$$

where πa^2 is the area in which the local autocorrelations are to be taken. From the definition of $G_i(\mathbf{R})$ it is evident that it may be immediately found if the positions of the dots in the pattern are known. If the local autocorrelation functions $G_i(\mathbf{R})$ for all the dots i in a cetrain region are added together, we will find densely packed clusters of delta functions in the areas corresponding to the separations of correlated pairs, while delta functions corresponding to the randomly distributed dots would be found uniformly spread over the rest of the area in which the local autocorrelation was taken. An algorithm could be found which would detect this clumping of delta functions. If we knew the properties required of the detection system, it should be possible to construct a maximally efficient system by judicious choice of such parameters as: the area πa^2 over which the autocorrelation is taken, the shape and size of the area to which the summation over i is restricted, the density of clumped delta functions required before detection occurs, and so on. This type of detection system relies on a large number of computations which could be performed in parallel, and thus is more practical for the eye, or analogue systems, than for digital computers.

The possibility that the human visual system uses autocorrelations is rather attractive[4]. Theoretical studies have indicated that pattern recognition (see ref. 5 for discussions and references to early work) and non-local, content-addressable memory[6,7] could be accomplished by using autocorrelation, and in psychological studies Julesz has demonstrated the importance of local correlations in both monocular and binocular vision[8]. We believe that the effect presented here provides additional experimental evidence that early steps of the human visual process may include the computation of local autocorrelations by

excitation of line detectors and the subsequent averaging of local autocorrelations by collective excitation of the columns in the visual cortex.

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DNA Ticketing Theory of Memory

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Contrary to usual opinions, it is possible to have a biochemically plausible theory in which memory is stored in coded form in the DNA of nerve cells.

From a biochemical point of view, one of the most puzzling features of long term memory is its permanence. At least in man, memories may survive for periods little less than the life of the organism. It does not follow rigorously from this that these long term memories are represented by biochemical alterations, but we consider it not unlikely that this is the case. Yet almost all cellular constituents are subject to turnover, even beyond the demands of cell division. Nerve cells do not normally divide in adult life and their DNA is generally considered to be metabolically stable but, as we shall see later, it is not usually regarded as a plausible candidate for the embodiment of the memory trace.

In principle it is possible to maintain differences between cells of the same genetic constitution, as a result of the existence of more than one stable set of concentrations of cellular components due to the non-linear equations controlling *m*RNA production, allosteric regulation of enzyme activity and perhaps other biochemical processes[1–5]. A theory of memory, based on such a model, has been developed previously by one of the authors[6,7] and is still considered by us to be perfectly plausible. In the present state of experimental ignorance, however, this should not prevent us from searching for other theoretical alternatives. Accordingly we describe in this article a new theory which seems to us to have an attractive simplicity and to afford some real possibility of experimental test.

The idea that memory is stored in coded form in the nucleotide sequence of DNA, specially synthesized for the purpose, seems highly improbable. About the only argument in favour of this notion (or the corresponding one involving RNA) is that it would provide the very high memory capacity of 10^{16}–10^{20} bits which the human brain has sometimes been supposed to possess[8]. There seems, however, no compelling experimental reason to believe that this capacity needs to be much more than 10^{11} bits, which is equivalent to the acquisition of about 30 bits a second continuously for a hundred years[9–11]. Indeed, if one accepts a figure of this order of magnitude and remembers that the brain contains some 10^{10} different nerve cells, one would rather argue that a memory coded by DNA or RNA would provide so great a potential capacity as to render it implausible on that ground alone. The final expression of such a coded memory must be presumed to be through the translation of nucleotide sequences into amino-acid sequences in proteins, and the existence of such a class of proteins would seem to provide an enormous difficulty for this theory. Furthermore, there is no evidence for the existence of any enzyme processes required for this kind of specific polynucleotide coding.

In spite of this, DNA does have an intuitive appeal as the depository of learned information as well as of genetic information. It is the one molecule which, apart from possible minor effects due to genetic damage and repair, we can be sure is present for the whole of the lifetime of the organism. And once we appreciate this point, we are impressed with the possibility that the peculiarity that nerve cells possess of not dividing has been devised so as to avoid disturbing the learned information which is somehow stored in their DNA.

The field of untrammelled speculation is, of course, limitless. In order to make a DNA theory of memory worth considering at the present time, we must show that it is biochemically reasonable in terms of current knowledge. So we now draw attention to the experimentally proven existence of enzymatic processes for modifying the nucleotide sequence of DNA. These chiefly involve the methylation of bases (usually cytosine) of which there are many different examples of apparently universal occurrence[12–16], although glucosylation has also been observed in the T-even coliphages. Of course, a base which has been modified in this way no longer belongs to the primary set of four, namely G, C, A and T. There may therefore occur certain consequences of the modification at the level of recognition of the DNA by enzymes or of its ability to combine with control elements such as inducers or repressors.

This leads to the suggestion that the physical basis of memory could lie in the enzymatic modification of the DNA of nerve cells. It might be worth looking to see if there are any unusual bases specific to nerve cell DNA, but in the absence of evidence to that effect, a plausible suggestion would be that the modification consists of methylation (or demethylation). We would propose that the methylation affects the rate of protein synthesis as, for example, by altering the affinity of an operator region for a