review articles

Visual experience and cortical development

H. B. Barlow*

The past twelve years have been exciting for those interested in the classical question, "How much do our visual capacities depend on innate developmental factors, how much on the moulding effects of visual experience?" Recent papers[1–4] have settled some issues, but may start new waves of controversy, and this is the occasion for writing this review.

THE oldest source of knowledge on visual development is clinical, for ophthalmologists know from their experience with amblyopia that one eye can become almost completely functionless from lack of use. This most commonly occurs when the two eyes interfere with each other, either because of strabismus (squint), or because one eye has a much greater refractive error than the other, and it can be partially prevented and reversed, at least in its early stages, by patching the good eye, thus forcing the use of the "lazy" eye that is threatening to become amblyopic. In 1963, Wiesel and Hubel[5] reported similar effects produced in cats by interfering with the normal binocular use of their eyes in the first month or two after they opened, and they discovered that the underlying change occurs in the cells of the primary visual cortex (area 17). In normal animals a high proportion of these neurones receive connections from both eyes[6], but if one eye is covered soon after birth, it makes connections with very few cortical neurones, almost all of which are then found to be connected solely to the eye which has been in continual use[5]. Furthermore, if a kitten is made strabismic by sectioning an eye muscle, or if joint usage of the eyes is prevented by covering each of them alternately, their neurones are found to receive input from one eye or the other, but very few receive input from both[7].

In their experiments Hubel and Wiesel found that covering both eyes had comparatively little effect on the cells of the cortex, suggesting a competitive element in the causation of the changes from monocular deprivation[8]. This was confirmed by Sherman, Guillery, Kaas and Sanderson[9] who made retinal lesions in one eye and closed the other. They found reduced effects of deprivation in the parts of the visual field that had no competing representation in the open eye. Hubel and Wiesel[10] found cells in the cortex of very young kittens, before the age of eye opening, that were connected to both eyes and appeared normal in other properties, and they reproduced many of these results in monkeys[11]. The apparent normality of cells in very young visually inexperienced animals, and also in binocularly deprived ones, led them to believe that the functional pattern of connections that underlies normal vision are laid down by genetically determined developmental processes. These can be interfered with or disrupted by abnormal visual experience in the "sensitive period" of the first few months of life[12], but they did not think that normal visual experience had any constructive effect in setting up the neural apparatus in the primary visual cortex.

These very important experiments with their suggested answer to the ancient dispute about the role of experience have naturally aroused great interest, and some controversy has arisen about both the facts and their interpretation. By now most of the facts discovered by Hubel and Wiesel have been confirmed by others, but there have been further advances that will be considered under four headings: ocular dominance and binocularity; specificity without experience; disparity selectivity; and the degree of modifiability of cortical neurones.

Ocular dominance and binocularity

The effects on the monocular and binocular connections of cortical neurones have proved reliably repeatable, but interesting additional knowledge has been gained by the technique of reversed suture[13–15], in which the originally closed eye is opened and the originally open eye is closed. It seems that an eye that has become disconnected can be persuaded to reconnect by renewed visual experience, provided that this occurs during the "sensitive period" and provided that the newly opened eye does not have to compete with an already connected eye that is in continued use. Thus experience can certainly have a reconstructive effect, and it is particularly interesting that the reconstructed connections are different in several ways from the previous connections that had been disrupted by the period of closure. The most striking evidence for this is provided by neurones that are in the process of being taken over by the newly opened eye but can still be driven by the other, previously open, eye. Some of these are quite unlike any cells found in normal cortex in having widely divergent preferred orientations for the two eyes, the difference sometimes being more than 70°, which is never found in neurones of normal cats and kittens. More information about this is obtained by examining the properties of the series of neurones encountered while advancing an electrode through the cortex, for the newly formed connections seem to develop a sequential pattern of orientational selectivity that is similar to the old pattern, but independent and often out of step with it[16].

The effects of deprivation are certainly easier to show than are those of experience, and a nice example of this was given by Olson and Freeman[17]. They found that 2–5 d of monocular vision had a profound effect on the ocular dominance histogram, but unexpectedly, it made no dif-

---

*Address: Physiological Laboratory, Cambridge CB2 3EG, UK

ference whether the animals had been reared in total darkness or in ordinary light before the monocular experience. For the deprived eye, several weeks of ordinary vision were undone in a few days of deprivation. Following reversed suture, however, one can see that experience encourages reconstruction, and one must suspect that normal joint usage of the two eyes may have some positive effect in establishing the normal orderly matching of the connections from the two eyes.

Specificity without visual experience

The properties of neurones in the adult primary visual cortex vary greatly, but it can be said of the vast majority that they selectively respond to only a very small proportion of the vast gamut of possible visual stimuli[6]. They are selective according to (1) the position of the stimulus, (2) whether lighter or darker than the background, (3) its direction and velocity of movement, (4) its size and orientation, and (5) the disparity or relative positioning of the image in the two eyes[18,19]. All are agreed that neurones with selectivity for (1), (2) and (3) occur in very young kittens without visual experience. Hubel and Wiesel also said there were many cells with the normal selectivity for the orientation of an extended object, as well as normal binocular connections, and it was on this basis that they concluded that all selectivity was innately determined. Barlow and Pettigrew[20] and Pettigrew[21] found clear-cut selectivity for the direction of movement of a stimulus in animals with no visual experience, and thought that the apparent orientational selectivity could be attributed to this directional selectivity. This seems to be wrong, for although many of the cells in very young animals are abnormal in their selective properties, there are about 25% (refs 2, 4) with the orientational properties of adult simple cells.

With regard to older, totally deprived, kittens Hubel and Wiesel again reported many normally selective cells, whereas Barlow and Pettigrew found a remarkable lack of them, and in this respect later work has supported them[2,4,22]. Even without the evidence from older kittens, however, it is now clear that all the types of selectivity of area 17 neurones, except that for binocular disparity, occur with no visual experience. The question whether they have as high a degree of selectivity as adults is still open, and there does seem to be a possible role of experience in "tuning up" their selectvities. This is a point of some interest because of the slow attainment of the high acuity characteristic of adults (*vide infra*).

Disparity selectivity

Most studies have followed Hubel and Wiesel in representing the binocular–monocular properties of cells in the visual cortex by "ocular dominance histograms". Cells are classified into seven categories varying from those excitable only through the contralateral eye (group I), through those excitable equally well by either eye (group IV), to those excitable only by the ipsilateral eye (group VII). Such histograms tell one the relative strengths of the excitatory inputs from each eye, but they tell one nothing about the way the inputs from the two eyes interact. This is important, not only because of possible facilitation, summation, or occlusion, but also because the input from an eye that does not excite a neurone can nevertheless inhibit it[23]. Thus the fact that ocular dominance histograms of very young kittens resemble those of normal adults does not mean that these animals handle binocular inputs normally. Pettigrew[21] studied this aspect of the problem in developing cortex and found a marked increase in the degree of selectivity for disparity with age and visual experience. Binocular interactions of young or inexperienced neurones occurred, but were much less precise and lacked the inhibitory component prominent in older and experienced neurones. The implication is that very young and totally deprived animals have normal ocular dominance histograms, but the critical inhibitory interactions of adults develop only with experience. This deserves repetition, especially in view of the contradictions about orientational selectivity, for it is again strong evidence for a constructive role of experience in forming the connections which combine the inputs from the two eyes.

Modifiability of other properties

A controversial problem is whether properties of the population of cortical neurones other than binocular connectivity can be affected by experience. Hirsch and Spinelli[24,25] and Blakemore and Cooper[26] reported dramatic results induced by exposing kittens to striped patterns, and Shlaer[27] produced some evidence that a prism in front of one eye deviates the receptive field positions of that eye. These results were extended in later reports[28,29] and since then modifications have been described as a result of rearing animals in stroboscopic light[30,31], in an evironment of moving stripes[32-35] and in a visual environment of dots without any stripes or lines[36,37]. Some of these treatments are claimed not just to modify the proportions of neurones of different types, but also to produce neurones with properties totally unlike any that are found in normal cortex. For instance Hirsch and Spinelli[25] reported units that seemed to have a receptive field exactly matching several bars of the grating the eye had been exposed to, and a close matching of receptive field to stimulus also seems to occur when kittens are raised in an environment of dots without lines[36,37]. The cortical units reported for these kittens would certainly be most unusual in normally reared animals.

Another example of abnormal units was described by Pettigrew, Olson and Hirsch[38] in the cortex of cats reared with one eye viewing vertical, the other horizontal, stripes, for they found units, like those described by Blakemore and Van Sluyters[14] following reversed suture, that had widely divergent preferred orientations in the two eyes. Again, when one eye is rotated surgically but not closed in young kittens[39], the position of the receptive fields at different points in the cortex stay unchanged relative to the retina. The orientations are not however normal: after correction for rotation the preferred orientations in the two eyes may be widely divergent in the few remaining binocular neurones. Once more we have evidence that coordinated binocular stimulation is needed to obtain coordinated developement of the appropriate selectivities.

Behavioural consequences of selective visual diets have also been described[40,41], though these are rather subtle and hard to observe compared with the effects of monocular deprivation[42,43]. There have been no reports of animals or humans with supernormal visual capacities following abnormal early visual experience, though this might be expected to arise according to some views. Some failures to obtain modification have been reported but hitherto these could be explained by differences of species, as when rabbits were used[44,45], or procedure, as when Maffei and Fiorentini[46] used vertical stripes of rather limited vertical extent and exactly regular periodicity.

At this point it seemed that, although most forms of selectivity were detectable in inexperienced cortex, they were also modifiable by subsequent visual experience. The report from Stryker and Sherk[1] however, has reopened the whole issue, for they made a careful and thorough attempt to reproduce the stripe-rearing experiment, following the procedures of Blakemore and Cooper, but they were unable to obtain significant results. The main points of difference were (1) an improved method of selecting the cells to be recorded from, aimed at obtaining more representative samples; (2) the use of a computer to control the stimulus and record the results, instead of hand-moved targets and subjective impressions; (3) ensuring that the experiment was done "blind", without the experimenters knowing the rearing conditions. Many people are likely to conclude that experimenter bias, reliance on subjective impressions, and an effectively very small sample of cells explain the dramatic findings of the previous experimenters, though it is not clear whether Stryker and Sherk hold this opinion. Against this conclusion is a brief study by Pettigrew, Olson and Hirsch[38] that was done blind, with computer control, and with positive results. There are further results, as yet unpublished, from Pettigrew's laboratory, and some of the papers already quoted contain incidental confirmation of the stripe-rearing effect (see also the note added in proof to this article). Obviously one must consider Maffei's and Fiorentini's[46] negative results again, but it seems to me unlikely that so many reasonably careful people should have been fooled so much of the time, and the clinical evidence to be described below establishes that orientational properties can be modified. It is therefore worth considering other possible explanations for the negative results.

The condition of the kitten when viewing the stripes is the natural point to probe. Was it alert? How much time did it spend looking up or down, and thus deviating the lines from their intended horizontal or vertical? What else did it see? Was it hungry, interested, rewarded? All these factors are hard to control, but may be expected to influence the results if the modification is anything like those occurring during normal learning. As a possible physiological explanation it is worth pointing out that Otten and Thoenen[47], studying the effect of preganglionic stimulation in inducing enzyme formation in sympathetic ganglia, had at one point great difficulty in getting reproducible results. This was traced to the glucocorticoid level in the blood at the time of induction: if the experiment was done in the morning, when the level was low, induction failed. Perhaps Stryker's and Sherk's cortical neurones ignored the stripes for the apparently irrelevant reason that they were exposed at the wrong time of day. Another possibility is that the sensitive period for orientational lability is earlier than that for ocular dominance, as Daw and Wyatt suggest for modifiability of directional selectivity[35]. There is also some evidence that dominance changes are slower than changes of orientation[29,48]. At all events the matter is unlikely to be accepted as closed and one must hope for more conclusive experiments in the future.

Evidence from psychophysics

These contradictory results go some way towards justifying the remark of one of the founders of cortical neurophysiology: "Recording from single units gives interesting results, but they are usually wrong". He was probably referring to other people's results, but he generously went on to say, "Visual psychophysics is dull but usually right". Even though psychophysicists do not necessarily share this confidence, it is worth trying to relate the neurophysiology to measurable psychophysical function, for there is new evidence that bears on the question of cortical modifiability. This actually lends quite powerful support to the single-unit results which have been called into question.

The main factor that limits the resolution of the adult visual system, as measured on straightforward tasks such as the visibility of a grating, is the finite size and separation of the elements of the retina. In the fovea of man this limit occurs at about 50 cycles per degree, and is set by the spacing of the receptor cells[49]. Away from the fovea, the minimum resolvable angle rises with eccentricity at the rate of nearly 0.5 min per degree, so that at 10° eccentricity only about 10 cycles per degree can be resolved. The limit here is probably also set by the retinal grain, in this case traceable not to receptors but to the separation of the ganglion cells that transmit details of the image centrally. In the cat it seems to be the size of the ganglion cells' receptive fields and their separation that limit acuity, even in the area centralis[50–52].

Clearly, if the fibres connecting eye to brain made very diffuse or scrambled connections one would not be able to resolve the image to a dimension set by the separation of their pick-up zones in the retinal image. These fibres must, therefore, make connections in the adult that are in some sense appropriate and orderly.

It is interesting that, for some tasks, the full resolution available from the retinal mosaic seems not to be used. Andrews, Webb and Miller[53] investigated the accuracy with which length comparisons could be made when the markers to indicate the lengths lay up to 5° from the fovea. After allowances have been made for eccentricity, the grain size that would be sufficient to enable this task to be performed was about 10 times larger, measured linearly, than that required for grating resolution. They made the interesting suggestion that this coarser mosaic is determined, not in the retina, but in the primary visual cortex, where it could correspond to the dimension, projected into the visual field, of the repeating subunit called a "hypercolumn" by Hubel and Wiesel[54,55]. This is the area of cortex, about 1 or 2 mm square, within which a complete cycle of ocular dominance is contained when moving across the cortex in one direction, and a complete cycle of orientational selectivity when moving in the orthogonal direction. It is thus a unit cell containing all orientation selectivities and all degrees of ocular dominance. Although the receptive fields of successive units isolated on moving an electrode across a hypercolumn show an average tendency to move across the visual field in the manner predicted from the overall mapping, an exact progression is not found[55,56]; the units are scattered around the expected positions in an apparently random manner, so it seems that the topographical orderliness for retinal position has been replaced by orderliness for orientational selectivity and ocular dominance. On the hypothesis being advanced, the output from a hypercolumn carries the information as to which hypercolumn is involved, but no information about position within it, and the consequent discarding of information causes the loss of resolution for the type of task Andrews *et al.*[53] studied.

Another task that suggests massive reduction of positional information is the counting of narrowly spaced lines that has been studied recently by Atkinson *et al.*[57]. They found that subjects started to make serious errors in counting even small numbers of lines when these are crowded close together: possibly the output from a hypercolumn can only signal "one" or "many", and for exact counting the lines in the target must fall on separate hypercolumns.

Thus, some tasks seem to be limited by the retinal grain, others by a coarser grain that may correspond to the hypercolumnar structure of the primary visual cortex. There are yet other tasks which at first seem to demand an even greater degree of precision than grating acuity. These are tasks like detecting the displacement of a vernier, the curvature of a line, or comparing the disparity of a pair of lines, and their common feature is that they require the detection of the relative positions of features in the stimulus that lie close to each other in the visual field. Since they are close, they could be within the area of visual field covered by one hypercolumn, and this type of information about relative position is not discarded, but jealously preserved. The precision with which these tasks can be performed is extremely high, of the order of a few seconds of arc, but Andrews, Butcher and Buckley[58] show that this is no higher than the accuracy expected from efficient use of the information in an image limited by the retinal grain. The improvement results from averaging, and simply corresponds to the fact that a mean is more precise than the individual measurements on which it is based. Thus there is no mystery about how the information required for the task gets to the brain, but a highly precise and orderly arrangement does seem to be required in order to extract it, and this is presumably one of the tasks of the hypercolumns in the primary visual cortex.

To summarise, then, psychophysics suggests there must be precisely ordered connections from eye to brain to preserve details down to the size of the retinal mosaic, and an even more precisely ordered arrangement within V1 for extracting the relative mean positions of features present in the image. But there is nothing impossible or implausible in these requirements, for hypercolumns are 1 or 2 mm across and contain some 10^5 cells, which allows plenty of scope for the operations demanded. The next step could be a reduction of positional information in the visual message brought about by retaining information about local pattern or relative position, but discarding absolute positional information, thus simplifying the picture by coarsening the mosaic to that of the hypercolumnar structure of V1.

Precise, orderly, and appropriate functional neurone–neurone connections are the essence of these operations. If they are made once and for all and are not modified or varied in potency by visual experience one would have a visual system whose performance depended exclusively upon its innate connectivity, a model sometimes referred to as the "hard-wired" cortex. A hardline advocate of hard-wiring will explain effects of experience exclusively in terms of disruption of innate connections, but arguments can be made on general grounds for a more constructive role. Platt[59] for instance held that a precision of a few seconds of arc could not possibly be laid down under genetic instruction, and would be hopelessly inflexible even if it could be. Andrews[60] argues that experience must be important in determining the metrical properties of perception, and Blakemore and Van Sluyters[2] suggest that experience ensures the alignment of the contributions from each eye to binocular receptive fields. Although these ideas are interesting they are not by themselves fully convincing. A teleological argument from the very existence of the sensitive period carries some weight, for it really is hard to believe in the survival value of a period of pure disruptibility, whereas a period of refinement and adjustment by experience would make good sense. At the moment there are no hard facts establishing such a role, but new evidence shows where the case for hardwiring is incomplete, and hints where visual experience may have its effects. Furthermore, recent experiments on the transfer of after effects support some of the concepts relating to cortical neurophysiology and psychophysics that have been discussed in this article.

Development of visual acuity

The neurophysiological argument for an innately connected cortex rests on the presence of selectively sensitive neurones in young, visually inexperienced kittens and monkeys. One could rule out any role of experience if such animals had full adult acuity, but this is far from the case. Acuity develops rather slowly in kittens[61,62], and does not reach its full adult value until quite late in the sensitive period. This could be a matter of removing other impediments to resolution, and as in all behavioural experiments one cannot be sure that the kitten is making full use of his sensory mechanisms, but the behavioural result is here supplemented by the study of cortical evoked potentials[61]. In humans too it seems that acuity is poor in young infants[63,64] and reaches a value comparable with that of the adult long after the system seems to be optically efficient.

One would like to know more about the effects of early binocular deprivation on visual capacities after sight is restored, for clearly if full acuity can ever be found in an animal that has been totally vision-deprived up to the moment of testing, this would rule out any important role for experience in performing resolution tasks. Clinical evidence on this is not very conclusive[65–67] and results on animals are not yet very extensive[43,61,62,67] but it does not seem that full visual capacities ever mature without considerable experience, and they often fail to do so if the experience occurs after infancy. If full performance cannot be achieved without experience, the implication is that normal experience aids cortical development and does not merely fail to hinder it.

Meridional amblyopia

The starting point of this whole enquiry was the fact that an eye can become blind when it is in competition with the other, functionally superior, eye, and the discovery that a similar condition occurs in cats and results from changes in the connections of cells in the primary visual cortex. It has long been recognised clinically that individuals with severe, long standing, astigmatism often fail to achieve good resolution in the affected meridian, even with the best correction of the optical condition. Testing the acuity of such subjects by generating interference patterns directly on their retinae has ruled out the possibility of a residual optical defect, and has now shown that this "meridional amblyopia", as it is called, is really of neurological origin[69]. Kittens reared wearing goggles containing cylindrical lenses have been shown to lack cortical neurones tuned to the blurred meridian, thus completing the cycle of evidence[70].

In spite of the difficulties in repeating the original stripe-rearing experiments, this clinical condition seems to establish beyond any reasonable doubt that the orientational properties of cortical neurones can be influenced by experience, though it does not of course establish that the changes are as described in the original neurophysiological experiments.

Stereo blindness

Strong neurophysiological evidence for a positive role of visual experience in setting up the visual cortex was given by Pettigrew's finding that cells narrowly selective for a particular disparity are not found in kittens that have had no binocular visual experience. The high incidence of anomalies of stereoscopic vision is therefore intriguing. Richards[71] estimates that 14% of the population lack the full ability to use binocular disparities to judge depths, and he reports interesting evidence that the stereo blind may fall into at least two classes—those who cannot use the convergent disparities of objects closer than the fixation point, and those who cannot use the divergent disparities of objects lying further away than the fixation point. Now phorias, or tendencies to strabismus, are common in infancy, and could cause deprivation of binocular stimulation. If the phoria is of the convergent variety, objects at the attempted fixation distance will have divergent disparities, but the system will tend to be starved of convergent disparities. Similarly those with a divergent phoria will be exposed to convergent disparities but will tend to be deprived of exposure to divergent disparities. The two forms of stereo blindness could thus be other examples of deprivation amblyopia.

Recent clinical evidence suggests that a week of monocular deprivation in infancy can lead to unilateral amblyopia in later life[72,73], so the sensitivity to deprivation is certainly very high in humans. The more subtle role of phorias in causing stereo blindness seems to be another potentially fertile field for correlating neurophysiological and clinical results.

Transfer of after effects

The view that proper stereopsis depends upon experience is supported by recent experiments comparing the transfer of after effects in normal adults and those who have seriously impaired stereoscopic vision, but have good monocular vision in both eyes, and no diplopia. From the cortical neurophysiology one might hazard a guess that they do not have disparity selective cells in area 17, and probably possess a population of neurones like that found after strabismus or alternating occlusion in kittens: that is, many cells monocularly dominated from each eye, but very few that receive binocular connections. Now there are a number of illusions such as motion after effects, tilt after effects, and distortions in the appearance and threshold modulation of gratings, that are commonly attributed to fatigue of cortical neurones; Maffei, Fiorentini, and Bisti[74] have recently shown that such fatigue does occur. Furthermore, such illusions normally transfer from one eye to the other, so that if the fatiguing stimulus is given to one eye, the illusion can be detected when that eye is closed and the other eye opened. Now if stereoblind individuals lack binocular neurones, such transfer should be impossible, and that is precisely the result that has been found[75–78]. In an ingenious application of this finding Hohman and Creutzfeldt[79] have concluded that the sensitive or labile period for development of binocular neurones in humans extends to the third year.

It is interesting that Ganz, Hirsch and Tieman[80] found normal interocular transfer of learned pattern discrimination in cats reared with alternating occlusion of the two eyes and therefore presumably possessing very few binocular cells in their primary visual cortex. This result does not conflict with the absence of transfer of after effects in such a cortex, because the tasks performed are so different. The persistence of transfer of learned discriminations presumably means that they were performed at a level where information as to which eye provided the image had been discarded.

Conclusions

The neurophysiological experiments that have helped so much in understanding the normal and abnormal development of the visual cortex are difficult and arduous to perform. The preparation involves several hours of surgery, and this is followed by a prolonged period of recording during which the animal has to be maintained lightly anaesthetised and in excellent condition. Worthwhile results are rarely obtained in less than 16 h, and recording often continues round the clock for 48 h or more. On top of this there are the frustrations and difficulties of maintaining cats in colonies that are notoriously liable to obliteration by epidemics. In view of this it is not the controversy but the agreement with Hubel and Wiesel's results that is surprising, and we owe to them most of our knowledge of the visual cortex as a precisely arranged structure laid down before visual experience, and thus innate. A good deal has, however, been added to their picture, and this shows it is not really established that all is innate. First, neither an inexperienced animal, nor an inexperienced neurone, has been shown to have the normal powers of resolution, and selectivity for disparity especially seems to require experience. Second, there is the evidence that experience after monocular deprivation causes the construction of a differently organised cortex. Third, Cragg[3] has recently published evidence showing that less than 1% of synapses in primary visual cortex develop before eye-opening, and hence there is, if Hubel and Wiesel are correct, an embarrassing difficulty in explaining what the other 99% do! But this merely underlines the disquieting fact that we do not fully understand what part area 17 plays in vision—itself another reason for caution in making pronouncements upon the relative importance of innate and experiential factors in creating the intricate mechanisms that underlie our visual capacities.

There is so much to be explained in the formation and adjustment of the connections between the 10^9 or so cells of our visual cortex that it is perhaps time to abandon the idea that nature and nurture are rivals at this task. That is too simple a view; instead one should suspect that they work in collusion, and seek how and where this may occur. Does the nervous activity from experience influence the survival and effectiveness of excitatory synapses? Does it exert its effect on the pattern of connections of an intrinsic inhibitory system that is established after the innate excitatory one? Or is there an interaction between nervous activity and the development process itself, as seems to occur in goldfish tectum[81]? The evidence reviewed here suggests that some of the answers are still waiting to be found in the mechanisms of the primary visual cortex that analyse details of the image and combine the information from the two eyes.

Note added in proof: Three new twists must now be added to the story on the effects of selective exposure. First, Hirsch and Levental[82] report more experiments in which kittens were reared wearing goggles of the type used in the original experiments of Hirsch and Spinelli[24,25]. In their new experiments the eyes viewed stripes within the goggles that were either left oblique for one eye and right oblique for the other, or horizontal for one eye and vertical for the other as in the original experiment. For the latter condition they confirmed, using more conventional neurophysiological techniques, that cells selective for horizontal orientations were almost all connected only to the eye that had viewed horizontal stripes, and similarly for the vertically orientated cells. There were a few cells which broke this rule by responding to verticals through the horizontally exposed eye and vice versa, but there were no cells at all that responded to oblique lines. The animals reared viewing obliques had many obliquely sensitive cells connected to the appropriate eye but also had many cells which responded to horizontal or vertical stripes through each eye. They conclude that genetic factors are sufficient to develop horizontal and vertical orientational selectivity, but that experience is required for the obliques.

The second twist is a note added to Stryker's and Sherk's paper[1] confirming these results for kittens reared with the horizontal/vertical goggles, using their more precise quantitative techniques. It is not yet known whether the differences between the results using goggles, and those from Blakemore's and Cooper's cylinders, is due to more effective control of the visual experience, or whether the conflicting experience of the two eyes in the goggles is the critical factor.

Finally, Grobstein and Chow[83] have reviewed their own work on the development of cortical specificity in the rabbit, and have also covered much of the material in this article. Their general conclusion is that individual sensory experience is a creative factor in establishing the functional organisation of the mammalian visual system, though they believe it affects the rate of development more than the end state reached.

[1] Stryker, M. P., and Sherk, H., *Science* (in the press).
[2] Blakemore, C. B., and Van Sluyters, R. C., *J. Physiol., Lond.*, **248**, 663–716 (1975).
[3] Cragg, B. G., *J. comp. Neurol.*, **160**, 147–166 (1975).
[4] Buisseret, P., and Imbert, M., *J. Physiol., Lond.* (in the press).
[5] Wiesel, T. N., and Hubel, D. H., *J. Neurophysiol.*, **26**, 1004–1017 (1963).
[6] Hubel, D. H., and Wiesel, T. N., *J. Physiol., Lond.*, **160**, 106–154 (1962).
[7] Hubel, D. H., and Wiesel, T. N., *J. Neurophysiol.*, **28**, 1041–1059 (1965).
[8] Wiesel, T. N., and Hubel, D. H., *J. Neurophysiol.*, **28**, 1029–1040 (1965).
[9] Sherman, S. M., Guillery, R. W., Kaas, J. H., and Sanderson, K. J., *J. comp. Neurol.*, **158**, 1–18 (1974).
[10] Hubel, D. H., and Wiesel, T. N., *J. Neurophysiol.*, **26**, 994–1002 (1963).
[11] Hubel, D. H., and Wiesel, T. N., *J. comp. Neurol.*, **158**, 307–318 (1974).
[12] Hubel, D. H., and Wiesel, T. N., *J. Physiol., Lond.*, **206**, 419–436 (1970).
[13] Wiesel, T. N., and Hubel, D. H., *J. Neurophysiol.*, **28**, 1060–1072 (1965).
[14] Blakemore, C., and Van Sluyters, R. C., *J. Physiol., Lond.*, **237**, 195–216 (1974).
[15] Movshon, J. A., and Blakemore, C., *Nature*, **251**, 504–505 (1974).
[16] Movshon, J. A., thesis, Univ. Cambridge (1975).
[17] Olson, C. R., and Freeman, R. D., *J. Neurophysiol.*, **38**, 26–32 (1975).
[18] Barlow, H. B., Blakemore, C., and Pettigrew, J. D., *J. Physiol., Lond.*, **193**, 327–342 (1967).
[19] Nikara, T., Bishop, P. O., and Pettigrew, J. D., *Expl Brain Res.*, **6**, 353–372 (1968).
[20] Barlow, H. B., and Pettigrew, J. D., *J. Physiol., Lond.*, **218**, 98–100P (1971).
[21] Pettigrew, J. D., *J. Physiol., Lond.*, **237**, 49–74 (1974).

22 Imbert, M., and Buisseret, P., *Expl Brain Res.*, **22**, 25–36 (1975).
23 Bishop, P. O., Henry, G. H., and Smith, C. J., *J. Physiol., Lond.*, **216**, 39–68 (1971).
24 Hirsch, H. V. B., and Spinelli, D. N., *Science*, **168**, 869–871 (1970).
25 Hirsch, H. V. B., and Spinelli, D. N., *Expl Brain Res.*, **13**, 509–527 (1971).
26 Blakemore, C., and Cooper, G. F., *Nature*, **228**, 477–478 (1970).
27 Shlear, R., *Science*, **173**, 638–641 (1971).
28 Spinelli, D. N., Hirsch, H. V. B., Phelps, R. W., and Metzler, J., *Expl Brain Res.*, **15**, 289–304 (1972).
29 Blakemore, C., and Mitchell, D. E., *Nature*, **241**, 467–468 (1973).
30 Cynader, M., Berman, N., and Hein, A., *Proc. natn. Acad. Sci. U.S.A.*, **70**, 1353–1354 (1973).
31 Olson, C. R., and Pettigrew, J. D., *Brain Res.*, **70**, 189–204 (1974).
32 Pettigrew, J. D., and Garey, L. J., *Brain Res.*, **66**, 160–164 (1974).
33 Tretter, R., Cynader, M. S., and Singer, W., *Brain Res.*, **84**, 143–149 (1975).
34 Cynader, M., Berman, N., and Hein, A., *Expl Brain Res.*, **22**, 267–280 (1975).
35 Daw, N. W., and Wyatt, H. J., *J. Physiol., Lond.*, (in the press).
36 Pettigrew, J. D., and Freeman, R., *Science*, **182**, 599–601 (1973).
37 Van Sluyters, R. C., and Blakemore, C., *Nature*, **246**, 506–508 (1973).
38 Pettigrew, J. D., Olson, C., and Hirsch, H. V. B., *Brain Res.*, **51**, 345–351 (1973).
39 Blakemore, C., Van Sluyters, R. C., Peck, C. K., and Hein, A., *Nature*, **257**, 584–586 (1975).
40 Vital-Durand, F., and Jeannerod, M., *Brain Res.*, **71**, 249–257 (1974).
41 Muir, D. W., and Mitchell, D. E., *Brain Res.*, **85**, 459–477 (1975).
42 Dews, P. B., and Wiesel, T. N., *J. Physiol., Lond.*, **206**, 437–455 (1970).
43 Ganz, L., in *Handbook of Sensory Physiology* (edit. by Held, R., Leibowitz, H., and Teuber, H.-L.), **8** (Springer, Berlin, in the press).
44 Mize, R. R., and Murphy, E. H., *Science*, **180**, 320–323 (1973).
45 Daw, N. W., and Wyatt, H. J., *J. Physiol., Lond.*, **240**, 309–330 (1974).
46 Maffei, L., and Fiorentini, A., *Science*, **186**, 447–449 (1974).
47 Otten, U., and Thoenen, H., *Proc. natn. Acad. Sci. U.S.A.*, **72**, 1415–1419 (1975).
48 Peck, C. K., and Blakemore, C., *Expl Brain Res.*, **22**, 57–68 (1975).
49 Campbell, F. W., and Green, D. G., *J. Physiol., Lond.*, **181**, 576–593 (1965).
50 Ikeda, H., and Wright, M. J., *J. Physiol., Lond.*, **232**, 34–35P (1973).
51 Bonds, A. B., *J. Physiol., Lond.*, **243**, 777–795 (1974).
52 Enroth-Cugell, C., and Robson, J., *J. Physiol., Lond.*, **239**, 30–31P (1974).
53 Andrews, D. P., Webb, J. M., and Miller, D. T., *Vision Res.*, **14**, 757–766 (1974).
54 Hubel, D. H., and Wiesel, T. N., *J. comp. Neurol.*, **158**, 267–294 (1974).
55 Hubel, D. H., and Wiesel, T. N., *J. comp. Neurol.*, **158**, 295–306 (1974).
56 Creutzfeldt, O., Innocenti, G. M., and Brooks, D., *Expl Brain Res.*, **21**, 315–336 (1974).
57 Atkinson, J., Francis, M., and Campbell, F. W., *Perception* (in the press).
58 Andrews, D. P., Butcher, A. K., and Buckley, B. R., *Vision Res.*, **13**, 599–620 (1973).
59 Platt, J. R., *Scient. Am.*, **202**, 121–129 (1960).
60 Andrews, D. P., *Q. Jl exp. Psychol.*, **16**, 104–115 (1964).
61 Freeman, D. N., and Marg, E., *Nature*, **254**, 614–615 (1975).
62 Mitchell, D. E., Griffin, F., Wilkinson, F., Anderson, P., and Smith, M. L., *Vision Res.* (in the press).
63 Atkinson, J., Braddick, O., and Braddick, F., *Nature*, **247**, 403–404 (1974).
64 Teller, D. Y., Mase, R., Baton, R., and Regal, D., *Vision Res.*, **14**, 1433–1439 (1974).
65 Gregory, R. L., and Wallace, J. G., *Q. Jl exp. Psychol.*, *Suppl.* **2** (1963).
66 Riesen, A. H., *Prog. Physiol. Psychol.* **1**, 117–147 (1966).
67 Ackroyd, C., Humphrey, N. K., and Warrington, E. K., *Q. Jl exp. Psychol.*, **26**, 114–124 (1974).
68 Sherman, S. M., *Brain Res.*, **49**, 25–45 (1973).
69 Mitchell, D. E., Freeman, R. D., Millodot, M., and Haegerstrom, G., *Vision Res.*, **13**, 535–558 (1972).
70 Freeman, R. D., and Pettigrew, J. D., *Nature*, **246**, 359–360 (1973).
71 Richards, W. A., *J. opt. Soc. Am.*, **61**, 410–414 (1971).
72 Awaya, S., Miyake, Y., Imaizaki, Y., Shiose, Y., Kanda, T., and Komuno, K., *Jap. J. Ophthal.*, **17**, 69–82 (1973).
73 Awaya, Y., Miyake, T., Kanda, T., and Shiose, Y., *Folia ophthal. jap.*, **25**, 270–281 (1974).
74 Maffei, L., Fiorentini, A., and Bisti, S., *Science* **182**, 1036–1038 (1973).
75 Movshon, J. A., Chambers, B. E. I., and Blakemore, C., *Perception*, **1**, 483–490 (1972).
76 Mitchell, D. E., and Ware, C., *J. Physiol., Lond.*, **236**, 707–721 (1974).
77 Warr, D., and Mitchell, D. E., *Vision Res.*, **14**, 731–735 (1974).
78 Mitchell, D. E., Rearden, J., and Muir, D. W., *Expl Brain Res.*, **22**, 163–174 (1975).
79 Hohmann, A., and Creutzfeldt, O. D., *Nature*, **254**, 613–614 (1975).
80 Ganz, L., Hirsch, H. V. B., and Tieman, S. B., *Brain Res.*, **44**, 547–568 (1972).
81 Yoon, M. G., *J. Physiol., Lond.*, **246**, 673–694 (1975).
82 Hirsch, H. V. B., and Leventhal, A. G., *Science* (in the press).
83 Grobstein, P., and Chow, K. L., *Science*, **190**, 352–358 (1975).

articles

New evidence regarding the Quaternary geology, archaeology and hominids of Chesowanja, Kenya

W. W. Bishop & M. Pickford

Department of Geology, Queen Mary College, University of London, UK

A. Hill

National Museums of Kenya, Box 40658, Nairobi, Kenya

Deposits yielding australopithecines, other fossil mammals and an industry recalling the Oldowan are succeeded by an Acheulian assemblage, overlain by flood plain silts with abundant spreads of later artifacts. The structure is dominated by an asymmetric anticline. Palaeoenvironments are reconstructed and the nature of the raw materials and of the tool makers themselves are briefly discussed.

CHESOWANJA is a Pleistocene fossil and artifact locality in the Northern Rift Valley of Kenya, about 1.5 km west of the track between Mukutan and Tangulbei, at 36° 12′ E, 0° 39′ N, and 8 km WNW of the point at which the Mukutan river gorge cuts through the Laikipia Escarpment. A diverse fauna includes the partial cranium of a robust australopithecine[1,2].

We report here results from more detailed geological mapping in 1973 and 1974. A remarkable sequence of artifact-bearing levels has been discovered together with a further hominid. The original geological interpretation has been shown to be in error, necessitating re-assessments of the local stratigraphic relationships, the palaeoenvironmental setting and the age of the sequence.

The area is important because the succession of rich artifact assemblages, from a relatively small and well defined region, spans a period probably well in excess of a million years. The associated geology and fossil fauna enable palaeoenvironmental contexts to be inferred for the various phases of hominid activity.

Geological succession

The local sequence of strata (Fig. 1) comprises four lithological units: the Chemoigut Formation, Chesowanja Formation, Karau Formation and Mukutan Beds. Formal descriptions of these and of the geology of the surrounding area will be published elsewhere.

The Chemoigut Formation consists of a sequence of silts and clays with intercalated horizons of coarse tuffaceous and pumiceous sandstone and fine conglomerate. There are calcareous bands and concretions, some of which are of algal origin, and thin flaggy and nodular calcrete horizons, several containing abundant root casts. There is also evidence of channelling. The observed thickness of the unit is ~ 25–30 m. The succession overlies red clays containing nodular calcrete, which elsewhere rest on very weathered ankaramitic basalts.

Artifacts have been found eroded out at a number of different sites from at least five horizons. Australopithecine remains come from the two highest levels. The fauna is listed in Table 1.