

How the Baby Learns to See: Donald O. Hebb Award Lecture, Canadian Society for Brain, Behaviour, and Cognitive Science, Ottawa, June 2015

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Hebb's (1949) book *The Organisation of Behaviour* presented a novel hypothesis about how the baby learns to see. This article summarizes the results of my research program that evaluated Hebb's hypothesis: first, by studying infants' eye movements and initial perceptual abilities and second, by studying the effect of visual deprivation (e.g., congenital cataracts) on later perceptual development. Collectively, the results support Hebb's hypothesis that the baby does indeed learn to see. Early visual experience not only drives the baby's initial scanning of objects, but also sets up the neural architecture that will come to underlie adults' perception.

Keywords: Donald O. Hebb, visual development, visual deprivation, visual perception, infants

In 1949, Donald Hebb published his now famous book *Organization of Behaviour: A Neuropsychological Theory* (Hebb, 1949). He presented a novel hypothesis about how the baby learns to see. A naive organism, be it infant or animal, he claimed first seeks out local points of maximum stimulation. When first presented with a triangle, for example, the naive will look at the corners and scan them repeatedly. At the same time, there will be motor tug from the lines abutting the corners, pulling the eye toward nearby corners, which are, in turn, scanned repetitively. The repeated scanning of local elements leads slowly to the development of a cell assembly for each element that is capable of functioning briefly on its own, as a closed system. The baby in our example slowly develops three diffuse cell assemblies, one for each corner of the triangle, while at the same time developing expectations about what will happen when he or she moves the eyes along a line connecting two corners. Those links become what Hebb termed *phase sequences*. At first, there is interference from other elements

in the visual field, such as the nest, or the wallpaper, or even mom, but if they occur irregularly at the same time as the triangle, they get eliminated from the cell assemblies forming for each corner and the phase sequences linking them. Eventually the combination of these cell assemblies and their phase sequences leads to the perception of a whole triangle.

This is an example of Hebb's mechanism for experience-dependent plasticity. The repeated scanning and sequencing leads to lasting cellular changes that allow more rapid firing, more stability, and larger percepts. It is an example of his general principle that "neurons that fire together, wire together": any two cells or systems that are repeatedly active at the same time are likely to become associated such that activity in one facilitates activity in the other. Hebb summarized his argument like this:

Perception depends on learning first to see the parts of an object clearly, a process involving a series of visual fixations, and proceeds from seeing, at first, an amorphous mass containing several foci (the corners), to seeing a distinctive figure at a glance. Even at this final stage we know the perception of the whole is dependent on eye movements for maximal clarity. (p. 101)

My doctoral supervisor, Phil Salapatek, arrived at Yale for his graduate training with Bill Kessen in the early 1960s when Hebb's theories were prominent but controversial, because they contradicted the prevailing views of both the Gestaltists and the behaviorists. The Gestaltists had used many perceptual primitives such as good continuation and common fate to explain why elements are grouped into a figure or relegated to the background. But their work was based on demonstrations with adults; Hebb argued that these perceptual primitives had originated from experience interacting with the world. The behaviorists advocated for learning, like Hebb, but their mechanisms of classically conditioned associations and reinforcement required that there be an external consequence for learning to occur, unlike Hebb's development of cell assemblies and phase sequences based only on repeated, sequential interaction with a stimulus.

I thank Dr. Terri L. Lewis, who has collaborated in almost all of the research I have done at McMaster University, as well as the many students, postdoctoral fellows, and faculty members who contributed to specific lines of research. I thank Dr. Henry Brent, an ophthalmologist at The Hospital for Sick Children, for steering me toward the studies of children treated for cataract and guiding my many years of research with those patients. Another ophthalmologist, Dr. Alex Levin, added guidance in later years of the project. I also thank the many funding sources that made the research possible: the U.S. National Institutes of Health, the Canadian Institutes of Health (and its predecessor, the Medical Research Council), the Natural Sciences and Engineering Research Council, the Social Sciences and Humanities Research Council, the James S. McDonnell Foundation, the Human Frontiers program, and the March of Dimes. Finally, I thank the patients treated for cataract who have volunteered for our studies for over 30 years. Without you, we would not have learned how the baby learns to see.

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Up until the early 1960s, tests of Hebb's ideas had been limited to experiments with animals and human adults, intact or with specific brain damage. Kessen's lab had both the insight that studies of newborns could be used to test Hebb's ideas and the technical savvy to gather those data. For his doctoral research, Salapatek built a system for measuring newborns' eye movements as they scanned a simple triangle. A typical result is in Figure 1. On the left are the fixations of two control infants while they scanned a blank field (the triangle was not presented to the baby but inserted as a reference for the analyses); on the right are the scans of two typical newborns presented with the triangle for almost 2 min (Salapatek & Kessen, 1966). The results look just as Hebb predicted: repeated scanning of the corners. Similar results were obtained for the scanning of other geometric figures (Salapatek, 1968).

When I arrived at Phil Salapatek's lab at the University of Pennsylvania in 1968, the first book he had me read was *Organization of Behaviour*, and together, we designed an extension of Phil's earlier studies to more natural stimuli, namely, the human face. We presented young infants with the faces of their mother, an unfamiliar female, and an unfamiliar male. Despite being presented for 75 s, the youngest infants (1-month-olds), spent the entire time fixating only a small part of the external contour—the chin for one baby, the hairline for another (Maurer & Salapatek, 1976; see Haith, Bergman, & Moore, 1977, for similar findings from newborns). Like the example in Figure 2 (left half), the infants seemed stuck on a local element of the external contour. In subsequent work, I discovered part of the reason: Babies have very poor peripheral vision. The newborn who is scanning the chin can barely make out the mouth, especially while attending to the chin, and cannot see the eyes at all. It takes about 2 months for the retina and cortex to mature sufficiently for peripheral vision to expand and for the baby to begin to respond to objects in the periphery while something else is in central vision (Atkinson, 1984; Maurer & Lewis, 1981). Then the baby shows evidence of

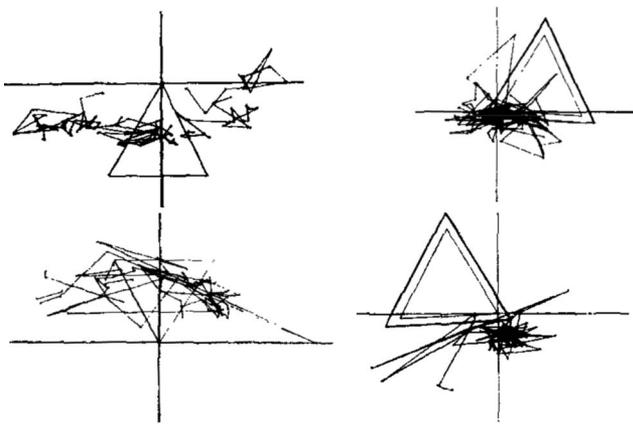


Figure 1. Typical scanning patterns for a newborn baby when presented with a blank field (left side) or triangle (right side) for 2 min. The triangle on the left side was not presented to the baby but added to facilitate scoring. As Hebb predicted, each baby scans a corner repetitively. Reprinted from *Journal of Experimental Child Psychology*, 3(2), Salapatek, P., & Kessen, W. Visual scanning of triangles by the human newborn. 155–167, Copyright (1966), with permission from Elsevier.

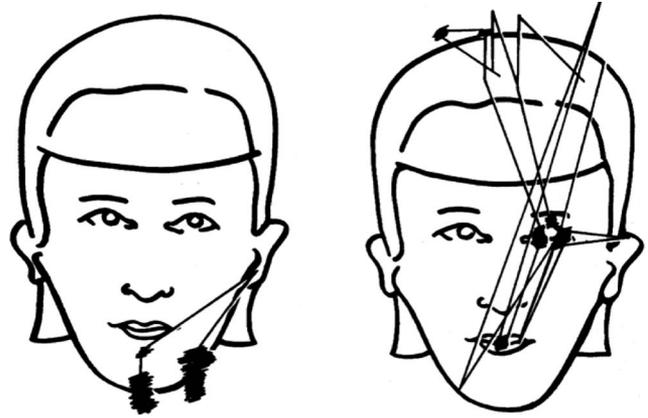


Figure 2. Typical scanning patterns for babies at 1 month (left side) and 2 months of age (right side). The stimulus was a live, three-dimensional face presented for 75 s. Even with natural stimuli like the mother's face, as Hebb predicted, young infants scan a piece of the contour repetitively.

connecting the Hebbian cell assemblies, scanning between the local elements as Hebb predicted. This is illustrated for a typical baby in Figure 2 (right half). Similar developmental patterns occur for triangles and other geometric figures (Maurer, 1983; Salapatek, 1975). As Hebb (1949) predicted, “figures do not act as wholes innately” (p. 19). The baby learns to perceive the whole from repeated scanning and interconnecting of its parts.

When I arrived at McMaster University as an assistant professor in 1973, I set up a lab to continue this interesting and theoretically important line of work. I reread Hebb's *Organisation of Behaviour* with my first graduate student, Terri Lewis, and we set out to chart the developmental changes in peripheral vision that may underlie the development of phase sequences. With the aid of the department's equipment shop, we designed and built a complex apparatus that allowed the recording of eye movements through stimuli presented on 2-ft \times 3-ft window screens. An infrared camera took pictures through each screen of the baby's pupil and the reflections thereon of five infrared lights that were invisible to the baby. The location of those lights changed systematically with the baby's fixation point. Cutting stimuli out of adhesive tape and pasting them onto the screens was time-consuming, the machine often jammed, and the screens had to be collected from a bin at the bottom after each baby and reinserted into the top from a ladder. But the worst part was scoring the data: The camera took four pictures per second, each of which had to be scored for the location of the center of the pupil and the reflections of each of the five lights. The resulting xy coordinates for each quarter second were punched onto a computer punch card. Nine minutes of data from one eye of one baby generated in excess of 2,000 cards, enough to overflow one box. Figure 3 shows some of the data I collected for my doctoral thesis. Next came running the cards through a program to determine the fixation point for each quarter second and analysing the patterns for each baby; all of that before analysing groups of babies at multiple ages. Interesting and theoretically important, perhaps, but a sure ticket to denial of tenure for me and a 10-year doctorate for Terri.

In rereading Hebb (1949), we noticed that he postulated that some aspects of perceptual organisation are present at birth, ap-



Figure 3. Studying eye movements before eye trackers. Each box of computer cards contains 9 min of data for one baby. Each card has been punched with the xy coordinates of the pupil of one eye and the reflections thereon of infrared lights. Those coordinates can be translated by a computer program into the location of the baby's fixations during 1/4 s of presentation of a stimulus. The boxes shown here are the data for my doctoral thesis at the University of Minnesota's Institute of Child Development. ©Charles Maurer.

parent even before the first visual experience. Figure/ground is an obvious example because the baby has to be able to pick out local elements of the figure to scan. He also mentioned (a) colour and (b) the orientation of lines. Hebb put colour on the list because of van Senden's reports on the retained colour vision of adults with developmental cataracts whose vision was restored. Hebb speculated that sensitivity to the orientation of lines is present without experience, or at least learned easily, based on his experiments with dark-reared rats, who could later discriminate orientation shortly after emerging from the dark. But learning to perceive the figure as a whole or to perceive the configuration of elements, he argued, required visual experience, of the type Salapatek and I had documented.

Fortunately for my career, a new method had just been described for measuring visual discrimination in young infants: infant-controlled habituation to criterion (Cohen, 1969; see also Horowitz, Paden, Bhana, & Self, 1972). In this procedure, an infant is presented with a visual stimulus, which remains on the screen for as long as the infant fixates it. Once the infant looks away, after a short interstimulus interval, the same stimulus reappears and the trials continue in this way until the time infant looks over three trials is less than 50% of the time during the first three trials. Then the infant is given a test that includes the now familiar stimulus and a novel stimulus. Longer looking at the novel stimulus than at the familiar stimulus is taken as evidence of discrimination (and short-term memory for the repeated stimulus).

With my early graduate students, Russ Adams, Myrna Martello, and Mayu Barrera, I ran with this technique to test Hebb's postulates about what is present in the newborn before visual experience. To measure sensitivity to colour, Russ Adams and I had to devise a technique that measured sensitivity to hue without spurious brightness cues to which the baby could respond. Had we

simply habituated babies to a grey square, then presented a red one, we would not have known whether the baby responded to the difference between red and grey or to the difference between two shades of grey, the one we presented and the one a colourblind individual perceives when looking at that red. Our solution was to get babies bored with looking at any shade of grey: During habituation, the baby saw 5 different shades of grey, intermixed in a random order. After habituation to criterion, the baby was shown two test stimuli: a novel shade of grey and a red square (chosen to be in the middle of the shades of the grey squares for an adult eye). Newborns' looking time stayed low for the novel shade of grey but increased significantly for the red square, thereby demonstrating some colour vision is present at birth (Adams, Maurer, & Davis, 1986). (These results do not allow us to conclude that the newborn sees red, as we do, just that the red square looked different from the grey square.) Subsequent experiments showed that newborns also discriminate grey from green and yellow and, at least by 1 month (the youngest age we tested), oblique lines slanted to the right from those slanted to the left (Maurer & Martello, 1980). But they failed discriminations involving configuration: After habituation to a schematic drawing of a human face, they failed to increase their looking when we switched to a drawing of a scrambled face (Maurer & Barrera, 1981). Only at 2 months did infants begin to show evidence of that discrimination. The results were as Hebb had predicted: initial detection of colour and discrimination of orientation but no sensitivity to configuration until visual experience is gained. The work evaluating Hebb was easy to publish, my students finished their doctorates (almost) on time, and tenure was assured.

Our work during this period also produced more direct evidence of perceptual learning. Using the habituation method described above, we discovered that, by 3 months (the youngest age we tested), infants can discriminate between the photographed faces of two unfamiliar women (Barrera & Maurer, 1981b) but also prefer to look at mom (Barrera & Maurer, 1981a): They fixate a photograph of her face longer than the photograph of an unfamiliar woman. Apparently, they have learned to visually recognise the mom through repeated exposure and scanning. In the discussion of these papers, we gloated that, by using a more sensitive technique, we had shown that babies have these abilities far earlier than reported previously in the literature. We went on to test with male faces and submitted a parallel paper to *Child Development* entitled "Recognition of the Father's Face by 3-Month-Old Infants." As with mothers, the infants had shown evidence for discriminating among male faces and preferring to look at a photograph of the father to that of an unfamiliar man. The manuscript was rejected! The reviewer noted that I had published a paper on female faces, another on the mother's face, and now I wanted to publish a paper on the father's face. "What next," the reviewer asked, "the grandmother's face, a cousin's face, and then the family dog?" I was stunned. But gradually, I began to see the reviewer's point: I was running through adult perceptual capabilities, testing them at younger and younger ages. This approach yields data on the starting point, which is important, but it is essentially antidevelopmental. It purports to study development in describing a superbaby, but says nothing about the mechanisms of stability and change. I gradually accepted the criticism but wondered what I could do instead: How does one study the mechanisms of change?

The answer came when I learned about a clinical project at The Hospital for Sick Children in Toronto, Ontario, Canada, involving a rare population of children born with congenital cataracts. This is a natural experiment providing direct evidence on the role of visual experience, or the lack thereof, on perceptual development. These children were born with dense, central cataracts in both eyes that blocked all patterned visual input until the cataracts were removed surgically and the eyes fitted with compensatory contact lenses. A comparison of their perceptual development to that of children with normal eyes allows deductions about the role of patterned visual input in driving developmental changes.

We began by studying acuity, the measure the parents and ophthalmologists were most anxious to know about. To do so, we used preferential looking grating acuity, a measure that takes advantage of infants' natural preference to look at something patterned like stripes. The stripes are paired with a grey square of the same mean luminance and are made smaller and smaller until the baby no longer shows a looking preference. The measure of acuity is the smallest size of stripes for which the baby looks at the stripes in preference to the matching grey square. Newborns with normal eyes respond to stripes that are just 30–60-min wide, whereas adults with normal vision can see ones smaller than 1 min (Dobson, Schwartz, Sandstrom, & Michel, 1987). In other words, newborns can see but their vision is about 60 times worse than that of an adult. Over the next 6 months, there is rapid improvement, but it takes about 7 years for grating acuity to reach adult levels (Elleberg, Lewis, Liu, & Maurer, 1999; McDonald, Ankrum, Preston, Sebris, & Dobson, 1986; McDonald, Sebris, Mohn, Teller, & Dobson, 1986). The results for infants treated for congenital cataract are similar: On the day they can first see, their vision is like that of a normal newborn, regardless of whether treatment was at 1 or 9 months of age (Maurer, Lewis, Brent, & Levin, 1999). In other words, the initial rapid development of acuity in infants with normal eyes is driven by visual experience; in the absence of that experience, it fails to occur.

But the visual nervous systems of infants born with bilateral congenital cataracts had not been dormant during the period of visual deprivation: After the first hour of visual experience, there was a significant improvement in acuity, to the level of a typical 6-week-old infant, and over the next few months, their acuity continued to improve faster than normal (Maurer et al., 1999) so that by the first birthday, almost all were within normal limits (Lewis, Maurer, & Brent, 1995). The parents and doctors celebrated: Treatment had been sufficiently early to allow complete recovery. Alas, their celebration was premature. We continued to measure the acuity of the patients longitudinally and found that at about age 2, it reached an asymptote, while the acuity of children with normal eyes continued to improve until about age 7 (Lewis et al., 1995). The result was a seemingly permanent deficit. This pattern is an example of a sleeper effect (Maurer, Mondloch, & Lewis, 2007b): Visual input early in life is necessary to set up, or preserve, the neural architecture for later learning. Studies of children who began life with normal eyes but who developed dense cataracts postnatally confirmed that visual input is necessary throughout the 7 years of normal development: A brief period of visual deprivation at any time during the first 7 years of life leads to a permanent deficit, although the size of the deficit is smaller, the later the onset of the deprivation. Surprisingly, visual deprivation between 7 years of age, when acuity is adult-like, and 10 years

of age also leads to permanent deficits: Visual input appears to be necessary to consolidate connections for a few years after they have formed and can support adult-like perception (Lewis & Maurer, 2009; Maurer, Mondloch, & Lewis, 2007a). These results indicate that the baby can pick out figures like large stripes from birth but learns to see finer stripes over many years postnatally.

In addition to acuity, we measured the effects of early visual deprivation on the ability to integrate details into a global percept, such as the perception of the direction in which objects are moving, an ability called *global motion*. You integrate visual information in this way whenever you see a flock of birds moving across the sky: despite many directions of local motion created by the flapping wings, you can easily perceive the direction in which they are heading. In the lab, global motion is tested with random dot kinematograms, first described by Newsman and Paré (1988). In our implementation, each dot on the screen moves in a predetermined direction for a limited time, then is reborn in a new location to move in that same direction. All the dots can have the same predetermined direction (e.g., 100% up) or only a portion of them (e.g., 50%) can do so, while the rest move in random directions. The task is to determine the direction of coherent motion (in this example, up). In our implementation, adults with normal eyes can do so when as few as 8% of the dots move up. Adults with a history of early visual deprivation from bilateral congenital cataracts require 40–50% of the dots to move coherently to detect their direction of motion (Elleberg, Lewis, Maurer, Brar, & Brent, 2002). This is another example of a sleeper effect: Infants with normal eyes demonstrate no sensitivity to the direction of motion before about 2 months of age (Braddick, Birtles, Wattam-Bell, & Atkinson, 2005; Wattam-Bell, 2003); yet infants treated for congenital cataracts by 2 months of age later have abnormal perception of global motion.

Studies of children treated for dense cataracts with postnatal onset yielded another surprise: Even when the deprivation began before the first birthday, sensitivity to global motion was completely normal (Elleberg et al., 2002). This is the same cohort in which acuity is abnormal when the onset of deprivation is before age 10. Both acuity and global motion depend on early input: In its absence, the system fails to develop normally later in life. Both have a long developmental trajectory: Acuity takes about 7 years to become adult-like; sensitivity to global motion improves until about 12 years of age (Hadad, Maurer, & Lewis, 2011). Yet the sensitive periods during which they can be damaged by early visual deprivation are totally different: 10 years for acuity and less than 1 year for global motion. These results illustrate that the critical period for damage cannot be predicted from the period of normal development. They also illustrate the value of longitudinal studies: Had we probed at just one time point, we would have drawn the wrong conclusions about the role of visual input in driving normal development.

Studies of other aspects of visual perception suggested that there are some that do not depend on visual experience: children treated for congenital cataracts developed a normal ability to detect whether a pattern is face-like (Mondloch et al., 2013), to discriminate the shapes of basic forms and facial features (Maurer, Lewis, & Brent, 1989; Mondloch, Robbins, & Maurer, 2010), to recognize facial expressions (Gao, Maurer, & Nishimura, 2013), and to perceive biological motion, that is, that a pattern of local movements comes from an animate organism (Hadad, Maurer, & Lewis,

2012). However, the normal sensitivity achieved by the patients is deceptive: The representation of facial expressions is not structured normally (Gao et al., 2013) and the neural representation of faces is abnormal (Grady, Mondloch, Lewis, & Maurer, 2014; Mondloch et al., 2013). For example, when adults treated for congenital cataracts look at faces, their accuracy and reaction times in discriminating faces from random patterns are (nearly) normal, but the event-related potentials recorded in their EEG potentials are highly amplified, with the degree of amplification correlated with the duration of the original deprivation from birth (Mondloch et al., 2013). It appears that it takes more-than-the-normal amount of neural activity to achieve normal performance. This could be in the normal neural network and/or in an alternative pathway.

Our early work supported Hebb's hypothesis that there are a few visual primitives, but that, to a large extent, the baby learns to see over the first few months by scanning the world. Our later longitudinal studies of children treated for cataract, indicated that the visual experience over those first few months is essential for setting up the neural architecture for later perceptual learning. When it is absent—when the cataracts are bilateral and congenital, there are many later sleeper effects, deficits that emerge later in development. Even abilities seemingly spared by the early deprivation can turn out to have abnormal neural underpinnings. The baby literally learns to see.

Like Hebb's writings, our empirical results led us to view development as a process with a few biological primitives, but mostly malleable processes that can be refined by sensory input to lead to an adult's specialized brain matching the individual's environment. By that point, the nervous system is no longer susceptible to damage from a period of visual deprivation. The corollary, we assumed, was that it can no longer be rescued from earlier insults. We recently discovered that corollary to be wrong. The participants were seven adults whom we had followed from the time they were diagnosed as infants with dense, central congenital cataracts in both eyes (Jeon, Maurer, & Lewis, 2012). We had documented many deficits resulting from their 3 to 10 months of early visual deprivation, including deficits in acuity, temporal and spatial contrast sensitivity, peripheral vision, stereopsis, perception of global motion, and perception of facial identity. Our therapy was an action video game called *Medal of Honour*. Built into a first person shooter game like this are the elements we theorized should optimise recovery: Difficulty is titrated to performance; it is fast-paced; the player needs to monitor both central and peripheral vision; it encourages learning in varied contexts, which should promote generalisation; it requires vision for action (acting with the joy stick on visual threats); and it is highly motivating. Such games also improve many aspects of visual performance in adults with normal eyes (Green & Bavelier, 2007; Li, Polat, Makous, & Bavelier, 2009). Our patients had a long day of pretests, played *Medal of Honour* for 10 hr/week for 4 weeks, and then repeated the pretests (Jeon et al., 2012). Overall, there were improvements in acuity, spatial contrast sensitivity, temporal contrast sensitivity, sensitivity to global motion, and perception of facial identity. There was no change in peripheral visual sensitivity or stereopsis, but the intervention lasted only 4 weeks and longer intervention or a better designed game may achieve even more.

In summary, early visual input is necessary for normal visual development and brain specialization, as Hebb postulated, but the brain remains sufficiently plastic for rehabilitation even in adult-

hood when it is too late to damage it by visual deprivation. That residual malleability likely would have surprised Donald Hebb, who expected that the system that had learned to see would not be changed so easily.

Résumé

L'ouvrage de Hebb (1949) intitulé *The Organisation of Behaviour* présentait une hypothèse novatrice selon laquelle le bébé apprend à voir. Le présent article résume les résultats de mon programme de recherche qui consistait à évaluer l'hypothèse de Hebb : premièrement, en étudiant les déplacements de l'oeil du bébé et ses capacités perceptuelles initiales et deuxièmement, en étudiant l'effet de la privation visuelle (par ex. en raison de cataractes congénitales) sur le développement perceptuel ultérieur. Collectivement, les résultats appuient l'hypothèse de Hebb suggérant que le bébé apprend effectivement à voir. Les expériences visuelles précoces entraînent non seulement le balayage initial des objets par le bébé mais aussi, établit l'architecture neurale qui servira de base à la perception de l'adulte.

Mots-clés : Donald O. Hebb, développement de la vision, privation visuelle, perception visuelle, bébés.

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