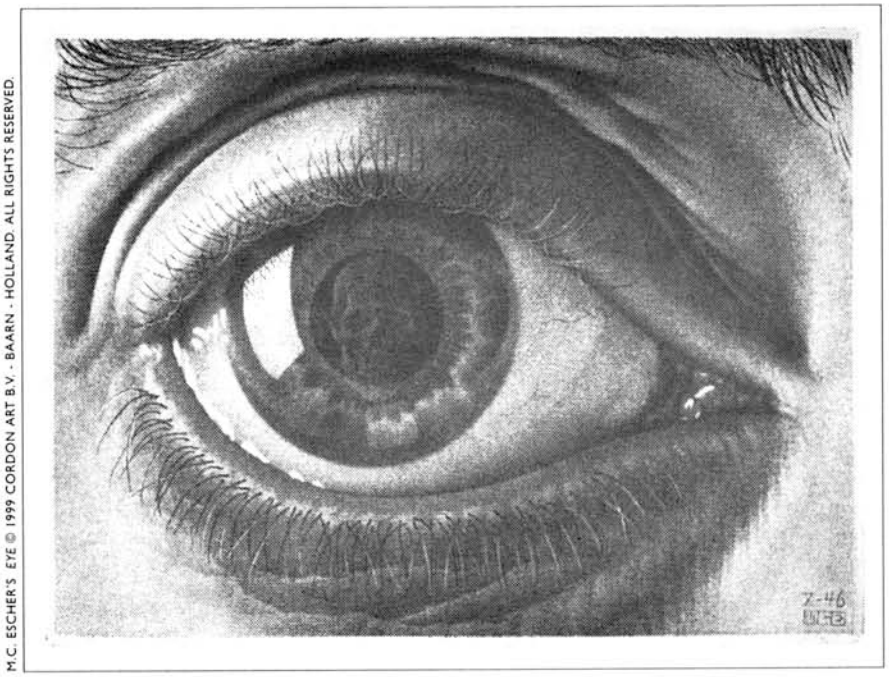

THE HOW AND WHY OF THE EYE

by Brian Freeman

We happen to use the eye for seeing—but, according to this anatomist, the fact that we are able to do so is not due to evolution or *design.*

"To this day the eye makes me shudder"—so wrote Charles Darwin to a botanist friend in 1860. Darwin called the eye an organ "of extreme perfection and complication" and struggled to comprehend how such an organ could have evolved through natural selection. To our day too, it remains hard to conceive how the human eye could have evolved. A random variation in the curvature of the cornea of an ancestral eye, for example, would cause a fuzzy image on its retina and thereby render the whole eye useless and an impediment to survival. To "evolve" a serviceable eye would require that any slight variation in a single parameter (e.g., corneal curvature) must be accompanied by a concatenation of simultaneous and harmonious variations in many other parameters (e.g., size of eye, size and curvatures of lens, refractive properties of lens, tension in lens ligaments, orientation of photoreceptors, etc.). Furthermore, such coordination would have to occur at every stage of the proposed evolution. However, so many integrated simultaneous variations would seem to be precluded by current evolutionary theory.

The great polymath, Hermann von Helmholtz (1821–1894), once remarked that one could hardly find a poorer optical instrument than the human eye: no optical designer could work with an inhomogeneous lens composed of living cells and a continuously varying refractive index.[1] And who, in their right mind, would think of designing a camera with a layer of light-absorbing structures directly in front of the film? Yet the eye is constructed with a layer of blood vessels embedded in the retina in front of the light-sensitive rods and cones. Even the vitreous body, that mass of jelly-like material through which the light has to pass between the lens and the deepest part of the eye (fundus[2]), is also an inhomogeneous medium since it contains remnants of embryonic structures. Finally, in addition to passing the retinal blood vessels, the light rays must traverse several layers of retinal nerve cells and their fibres before exciting the rods and cones. From the viewpoint of imaging technology, the rods and cones are designed for the wrong side of the retina!

In the absence of assistance from evolutionary theory or design theory, how is one to comprehend the human eye?

For a start, we must dispense with the notion that the eye is an organ "*designed* for seeing". Such a claim is mere teleology, and is therefore unscientific: there is simply no way of proving or disproving such a claim using rational scientific methods. In embryology and anatomy, teleological thinking (i.e., the notion that outcomes guide development) leads to many paradoxes, such as Helmholtz's conundrum concerning the eye. In actual fact, we cannot comprehend the development, the anatomy, or the function of any organ whatsoever, by making a claim about *why* the organ developed, based on *how* the organ happens to be used once it has developed.[3]

The reality of development is more like this: a tiny organ (let us call it organ "X" to avoid any preconceptions) arises gradually in some region of the human embryo. As it grows, this dynamic organ acts on other parts of the embryo, causing other structures to

arise in reaction. The sequence of alternating developmental actions and reactions starting from "X" results in an ensemble of organs known as the eye with its surrounding components (orbital adnexa) such as eyelids, tear gland, eye muscles, bones of the eye-socket, fat behind the eyeball, etc. During normal development, by definition, all components of the ensemble at all stages must be totally integrated and functioning harmoniously; the slightest discord would produce an abnormal eye. Naturally the embryo has no prior knowledge of how this ensemble will be used after birth. The only accurate claim is that, as a result of its development, this coordinated ensemble has some parts more-or-less transparent, has some parts movable at will, has parts that allow the external world to be imaged, parts with much black pigment (melanin) to absorb stray light, parts that are excited by electromagnetic radiation of certain wavelengths, and so on.

"How is one to comprehend the human eye?"

In other words, eyes do not develop in order to see; rather, sight is one of the consequences of the development; the ability to cry is another; winking at friends yet another; etc. Nothing in the ensemble of structures associated with the act of seeing should be dismissed as being of "secondary" importance compared to a presumed "main role" of seeing (as implied by the term orbital adnexa). For example, eyes without eyelids or tear glands would soon become useless because of ulcerating corneas; in stationary eyes without eye muscles, the stabilised images formed on the retina would fade quickly from consciousness.

So to understand the how and the why of the whole eye, we need to identify the first hint of the eye-to-be in the form of the organ "X", and then to trace its gradual development. Let us therefore look briefly at the development of the head of the human embryo to see how it helps us to comprehend the puzzling anatomy of the eye.

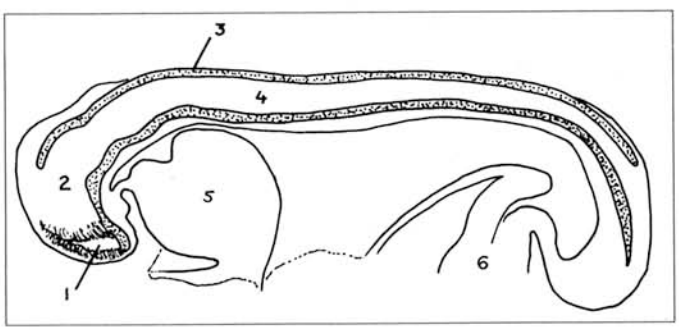

Fig. 1: Sketch of right half of a human embryo about 2 mm long, after reconstruction and cutting through the midline (median plane), viewed from left. Stipple indicates cuts through the roof and floor of neural tube. 1 right eye field (precursor of eye), 2 right wall of neural groove at head end (in vicinity of cranial neuropore), 3 ectoderm (skin) covering neural tube, 4 lumen of neural tube, 5 heart, 6 connecting stalk to developing placenta (reconstruction by Heuser).

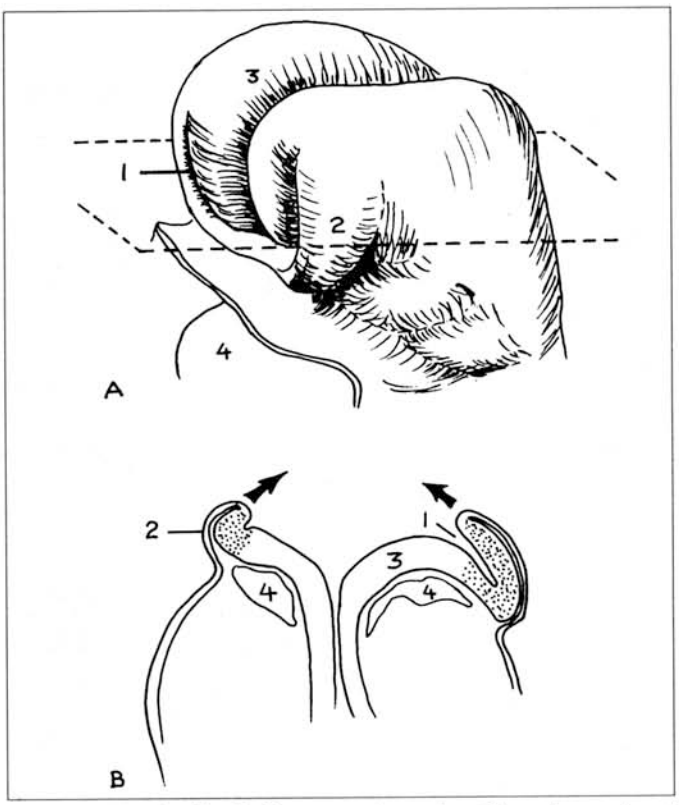

Fig. 2: a) Sketch of head of human embryo about 3 mm long, viewed from left oblique. 1 right optic groove, 2 ectoderm covering left eye (precursor of lens), 3 right wall of neural groove in vicinity of cranial neuropore, 4 heart.
b) Sketch of horizontal section through head of same embryo, cut in plane marked by dotted line in a). Stipple indicates precursors of optic vesicles (note asymmetry in size of eye structures—symmetry does not exist during embryonic development). 1 right optic sulcus, 2 ectoderm (skin) covering precursor of left optic vesicle, 3 right wall of neural groove, 4 large veins of head. Arrows indicate direction of closure of margins of cranial neuropore.

THE NEURAL AND OPTIC GROOVES

Two to three weeks after conception, when the embryo is less than 2 mm long[4], a midline groove (neural groove) develops along the skin (ectoderm) of its back. The whole back of the embryo, including the groove, is bathed by amniotic fluid contained in a tense membrane (amnion). The neural groove arises partly from a longitudinal buckling that takes place as the ectodermal sheet of embryonic skin expands in surface area against the resistance of the tenser amnion, which is joined to the sheet's perimeter. Either side of the midline, the walls of the neural groove rise up to make two neural crests. The cells comprising the walls of the neural groove (neurectoderm) multiply faster than any other cells in the embryo and so the walls thicken, as well as increase their surface area. The walls soon close over, initially in the region of the embryo's neck, to form a tube (neural tube). The neural tube itself becomes covered by skin along the embryo's back; the cells in its walls later become the cells of the spinal cord and brain, and the fluid contained in the lumen of the tube

becomes the precursor of the "water" in the ventricles of the brain and the spinal canal (cerebrospinal fluid).

At the head or cranial end of the embryo, the walls of the neural groove do not close for some time. The longitudinal slit-like opening found here is called the cranial neuropore. Well before the cranial neuropore closes, the **precursor of the eye** (the eye field) can be identified on each side as a shallow depression in the surface of the right and left walls of the neural groove *(fig. 1)*. Each depression could be considered to be the equivalent of our organ "X" for the eye.[5] The wall of the depression thickens so that the shallow concavity becomes squeezed into a groove (optic groove or sulcus). Viewed from the front of the embryo, each optic groove appears like a crease arising from a local, outward buckling of each wall of the still-open neural groove *(fig. 2)*.

"Naturally the embryo has no knowledge of how this ensemble will be used after birth."

The optic groove projects laterally and its wall lies directly below the skin (ectoderm) of the embryo's head *(fig. 2b)*. The two optic grooves cease to be visible from outside the embryo after the cranial neuropore closes due to multiplication of the cells in the walls of the neural groove. On account of this closure, the precursor of each eye ("X") has now developed into a kind of cul-de-sac of the fluid-filled brain tube (strictly, a diverticulum of the forebrain lumen). This bubble-like sac is called the **optic vesicle** *(fig. 3)*. The optic vesicle remains joined to the wall of the neural tube by a tubular stalk (optic stalk), and encloses a fluid-filled lumen (optic ventricle) which is continuous with the ventricle of the brain.

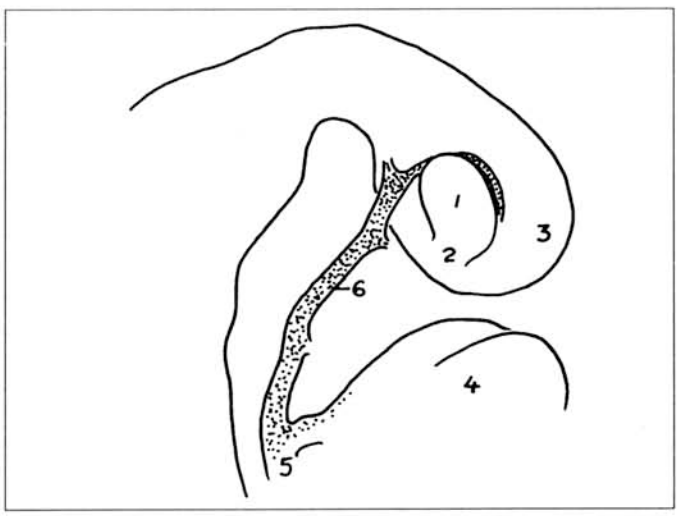

Fig. 3: Sketch of brain and blood vessel to head (stippled) in an embryo 4.2 mm long, viewed from right (ectoderm transparent). 1 right optic vesicle, 2 region of optic stalk, 3 forebrain, 4 heart, 5 aorta, 6 internal carotid artery.

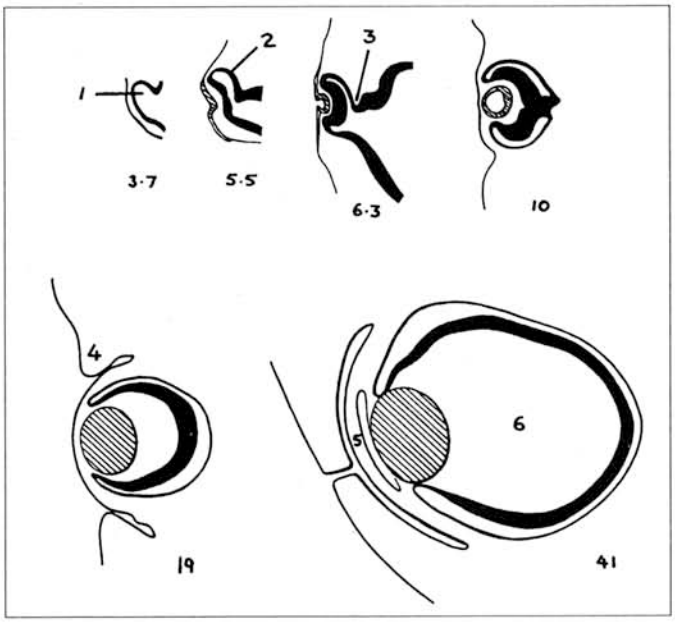

Fig. 4: Sketches of vertical cuts through developing eyes from embryos 3.7, 5.5, 6.3, 10 & 19 mm long, and from a fetus 41 mm long, respectively. Developing lens hatched, sensory retina and pigment epithelium black. 1 lumen of optic vesicle (optic ventricle), 2 outer wall of optic cup (precursor of pigment epithelium), 3 region of optic stalk, 4 upper eyelid, 5 cornea bordering pool of aqueous humour in anterior chamber of eye, 6 vitreous body. Note eyelids have almost fused in 41 mm fetus.

The optic vesicle grows in surface area and bulges outwards beneath the embryo's skin. Since the optic stalk does not grow so fast, it becomes relatively narrow. The growth of the wall of the optic vesicle is not uniform in all its parts. Initially, that part of the wall lying directly under the skin tends to thicken faster than it grows in surface area, whereas that part of the wall lying further from the skin remains thin and displays greater surface growth. Where the wall of the optic vesicle changes from thick to thin, especially superiorly, many cells are squeezed out to become the future connective tissue and muscle cells of the eye (optic mesectoderm—the precursor of parts of the uvea, cornea, sclera, extra-ocular muscles, etc.). The external surface of the optic vesicle sheds so many cells in such a short time, that it takes on a spiky appearance and has been likened to a hedgehog. Later its contour will become smooth again. A branch of the internal carotid artery to the embryo's head lies near the inferior surface of the optic stalk and supplies nutrients preferentially to that part of the wall of the optic vesicle directly under the skin. In this way, blood vessels contribute to thickening growth. This asymmetric growth of the vesicle causes its shape to change: its lumen narrows to a slit and the outer wall approaches the less superficial wall. As it thickens, the outer wall invaginates, so-to-speak, into the lumen of the optic vesicle and the resulting structure is called the optic cup *(fig. 4)*.

THE OPTIC CUP

From now on, the optic cup will comprise two concave walls or sheets of cells that grow in surface area at different rates, thereby coming closer and closer together. The thicker wall facing the ectoderm becomes the **sensory part of the retina,** while the thinner, outer wall becomes the **pigment epithelium** of the retina. The outer wall is surrounded by embryonic connective tissue (optic mesectoderm) which provides a medium through which nutrients can percolate (the precursor of the middle layer of the eye, called uvea). Later, blood vessels arise in this connective tissue bed and supply nutrients directly to the pigment cells. The folded-over rim between the two walls will form the iris and the aperture within the rim, the **pupil.**

"The first blink is an exceedingly slow movement, taking days."

The pigment epithelium is so named because it contains a black pigment (melanin). It is the first non-transparent tissue to appear in the human embryo. As the outer wall of pigment epithelium approaches the sensory retina and the lumen (optic ventricle) becomes obliterated, the pigment cells are able to nourish the sensory retina directly. This occurs because the pigment cells export nutrients absorbed from the neighbouring connective tissue (uvea) to the cells of the sensory retina. The unique feature of these movements of metabolites is that, here, a single epithelium both imports nutrients from an underlying connective tissue and simultaneously maintains a nutritional supply to a neighbouring epithelium. The intensive supply of nutrients enables the sensory retina to accelerate its areal growth. By virtue of this growth, the sensory retina now becomes the "prime mover" or engine for the subsequent development of the eye and most of the extra-ocular structures. The metabolic connections between sensory retina and pigment epithelium continue throughout life: a detachment of the one from the other (no matter whether due to trauma, or to a blister-like accumulation of fluid at the site of the original optic ventricle) leads immediately to blindness (scotoma).

The optic cup, which has developed from the optic vesicle, is like a tiny hand that enables the brain to grasp, as it were, a piece of embryonic skin within its fingers: this piece of skin is the precursor of the lens *(fig. 4)*. At first the **lens** is a patch of thickening skin (ectodermal lens placode) confined within the inner margin of the optic cup. Its thickening too, is underpinned by a preferential supply of nutrients from the same branch of the internal carotid artery (hyaloid artery) that contributed to the differential thickening of the wall of the optic vesicle (the precursor of sensory retina). The cells in this "patch" prosper and multiply close to their nutrient supply, while the surrounding, thinner skin spreads away: the shape of the lens placode thereby changes to a depression (lens pit). Eventually, the pit transforms to a lens vesicle that soon loses its connection with the skin. The ectoderm that seals over the lens vesicle becomes the external surface of the cornea. Only when the wall of the lens vesicle thickens, by virtue of an intensive uptake of nutrients and a simultaneous release of its watery by-products of metabolism, will the vesicle become the crystal-clear lens of living cells that later serves to regulate the refraction of light into the eye.

The watery by-products of lens growth accumulate in the adjacent superficial connective tissue under the curved ectoderm of the future cornea, so forming the anterior chamber of the eye containing a fluid, the **aqueous humour** *(fig. 4)*. The connective tissue cells that remain between the pool of fluid and the corneal ectoderm, are stretched and flattened by the pressure of the accumulating aqueous humour on the one hand, and the tension and surface growth of the ectoderm on the other. Collagen is "wrung out", so to speak, from the cytoplasm of these stressed connective tissue cells. The sheets of collagen fibrils that align along the lines of tension contribute to the bulk of the **cornea** (substantia propria corneae). When sufficiently hydrated, the collagen fibrils in these curving sheets are so spaced that, collectively, they can transmit electromagnetic radiation of certain wavelengths and change its direction (refractive power of a transparent cornea). Under conditions of insufficient hydration, the interspacing of the collagen fibrils decreases and corneal opacity results. As the eyeball grows, other connective tissue cells lying external to the uvea also become simultaneously flattened in a radial direction and extended circumferentially. Sheets of collagen are also laid down here, but this collagen, with a different packing density of fibrils and a different degree of hydration to that in the cornea, becomes the tough, opaque **sclera** ("white" of the eye) that serves to protect the uvea.

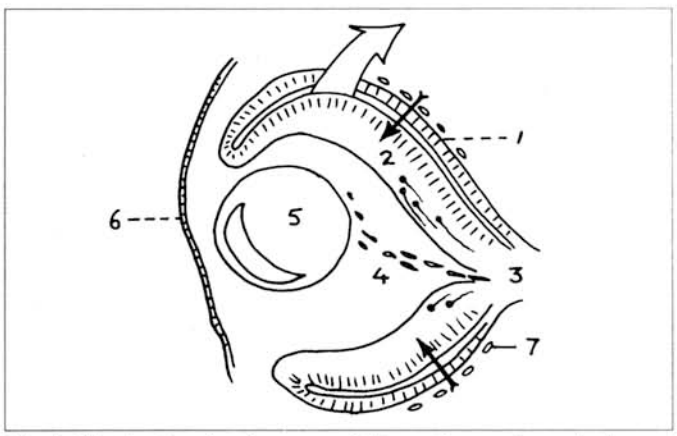

Fig. 5: Sketch of section from eye of 13 mm long embryo. 1 pigment epithelium, 2 sensory retina (with bodies of retinal ganglion cells and their neurites indicated under 2), 3 optic stalk, 4 precursor of vitreous body (with branches of hyaloid artery supplying lens indicated in 4 above), 5 lens vesicle, 6 ectoderm of cornea, 7 blood vessel in uvea. Open arrow indicates direction of retinal flattening, solid arrows indicate movement of nutrients into sensory retina.

THE RETINA AND THE OPTIC NERVE

It is worth remembering that the wall of the optic vesicle is an extension of the neural tube. The processes[6] of the cells in the wall of the vesicle are aligned radially in a direction towards the nourishing connective tissue (optic mesectoderm), that is, towards the periphery of the optic vesicle. As the optic vesicle transforms into the double-walled cup shape, the cellular processes that were originally in the vicinity of the overlying skin now lie in the inner wall of the cup, directed radially towards the back of the lens. Then, as the optic cup grows larger, its walls flatten, just like the re-opening of the fingers and palm of the hand that previously grasped the little ball of lens cells. Now an early optic cup has a vertical diameter of, say, 0.4 mm (in a 6 mm long embryo) whereas the adult eye has a vertical diameter of 23.5 mm. Over the course of its development to adulthood, the retina has grown enormously in surface area and progressively flattened, but its thickness has scarcely changed. The relative constancy of the thickness of the retina is a remarkable feature of ocular growth.

"The optic nerve connections simply elongate, eventually to their adult length."

The flattening of the retina results in a striking change in the orientation of the fibrous processes of those cells in the innermost part of the retina. The bodies of these cells are dragged in the direction of retinal flattening, which is more or less at right angles to the processes of the deeper cells that retain a radial orientation *(fig. 5)*. When the innermost cell bodies first become displaced, they leave their processes (neurites, or axons) trailing behind. At first glance, it appears as though it is the fibrous processes themselves that are growing actively away from the cell bodies. However in reality, the tips at the ends of these processes remain virtually fixed near that part of the optic cup where the rate of change of retinal curvature is minimal. Initially, this corresponds to the place where the optic stalk attaches to the eye. Thus, a layer of **nerve fibres** arises along the inner surface of the sensory retina, with tips remaining closer to the optic stalk and processes fanning out from here to their passively displaced cell bodies (retinal ganglion cells with their neurites). Soon the tips of these nerve fibres will start to advance actively along spatially ordered channels that have arisen in the interstices of both the sensory retina and the inner wall of the optic stalk (guidance pathways for growth cones). These channels contain fluids which, we must assume, convey nutrients to the now actively growing tips of the optic nerve fibres. Just as rootlets of plants suck themselves towards sources of nutrition, so too the tips of the optic nerves make metabolic connections with other cells in specific regions of the brain, and grow toward these regions. The distances are relatively short, e.g., in an embryo 13 mm long, the eye is only 1 mm or so away from the nearest destination of its nerve fibres. As the brain and head grow, the optic nerve connections simply elongate, eventually to their adult length of between 9 and 11 cm between the eyeball and their terminals in the thalamus (lateral geniculate nucleus).

In the retina, the innermost cells are the first nerve cells to mature (retinal ganglion cells). In contrast, the photoreceptors which lie in the deepest part of the sensory retina adjacent to the pigment epithelium, are the last cells to mature. This explains why a light-ray must first traverse layers of nerve fibres and cell bodies before reaching the layer of rods and cones (outer segments of photoreceptors). The place where the nerve fibres pass from retina into optic stalk will have no photoreceptors, and so will become the blind spot of the fundus.

THE POSITION OF THE EYES

The eyes form initially at the sides of the embryo's brain, i.e., facing laterally *(figs 2, 3)*. After the cranial neuropore closes, the growing brain pushes into the skin of the forehead region (frontal lobes). The connective tissue below the forehead and between the two eyes is compressed transversely and becomes taut, thereby tethering the eyes. This tense tissue constitutes a ligament that is stretched across the face at the bridge of the nose (interorbital ligament). The growth of the brain causes the forehead and mid-face to broaden. The widening mid-face, in combination with the anchoring of the nasal aspect of the eyes, forces the eyes into a more and more frontal position *(fig. 6)*. Eventually, the interorbital ligament will become the membrane covering parts of the frontal and nasal bones of the skull (periosteum in the vicinity of the nasion).

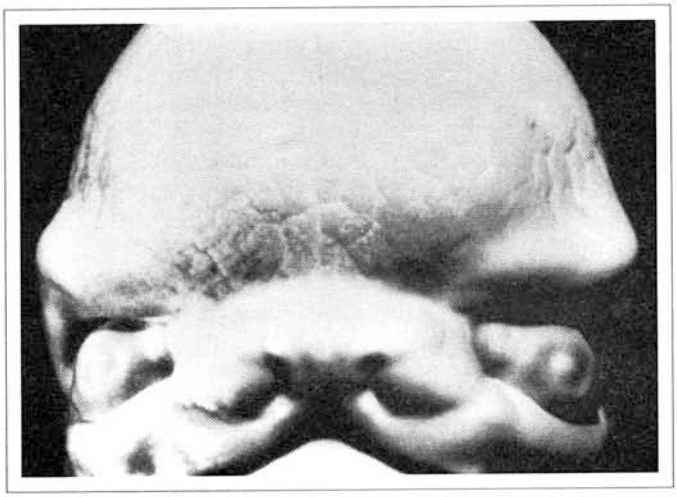

Fig. 6: Forehead and mid-face of a human embryo, 16.2 mm long, 6.5–7 weeks after conception. Note furrow between eyes (site of interorbital ligament), corneas and initiation of skin folds above and below each eye (eyelids). At this age the nose is broad and flat, the nostrils are small, and the mouth curls down at the corner.

THE EYELIDS AND BLINKING

Initially, there are no eyelids: the skin of the cornea continues directly into the skin of the face. However, the interorbital ligament fans out laterally either side of the bridge of the nose into the skin both above and below each eye. The slowly increasing tension in the ligament causes skin folds to appear above and below the eye, in the same way that tension in cloth or rubber causes folds. These folds are the first hint of the **eyelids** *(figs 4, 6, 7)*. Subsequently, the tension between the eyes becomes so great that the folds are dragged slowly across the cornea, until they fuse together during early fetal development *(figs 4, 8)*. This is a non-muscular closure of the eyelids, due principally to the growth of the brain and the mutual tethering of the nasal aspect of the eyes.

As a skin fold is slowly transforming into an upper eyelid, the cells beneath the skin are stretched in a direction perpendicular to the edge of the fold, i.e., almost at right angles to the line of tension in the interorbital ligament. As a consequence of their extension, these cells are transformed into muscle cells whose subsequent hardening ("contraction") during the continual growth of the head, will lead to a fixation of the upper eyelid (action of levator palpebrae superioris muscle). Thus, the previously closed eyelids will slowly "unfuse" (dehisce). The widening of the gap between the eyelids (palpebral fissure) slowly stretches other cells lying beneath the skin into an annular muscle encircling the fissure (orbicularis oculi muscle). When this muscle tenses, the eyelids will tend to close—this is the first **blink** of the embryo. The movement is due to growth, not muscles. It is an exceedingly slow movement, taking days, and it is imperceptible except through an accurate analysis of successive stages of embryonic anatomy. Nonetheless it is a movement, with its own special dynamics: it could be called a growth-blink. The sequence repeats itself, only faster, each time leading to the development of more powerful antagonistic muscles. Finally the movements become so rapid that they can be detected as **blinking** during ultrasound scans of fetuses around the 26th week of pregnancy.

In the development of the capacity to blink, we see one of many examples of a reversal between an embryonic function and an adult function: embryonic cells must first be stretched by the growth-activity of surrounding cells and cellular ensembles, before they can develop into muscle cells capable of shortening. Another example: embryonic cells must first be stretched in one axis and compressed from the sides before the collagen "wrung out" of them is strong enough to resist further deformation (development of cornea and sclera, ligaments and tendons). At any stage, whether embryonic or adult, the capacities of a human organism are the direct consequences of the totality of prior development. Without a shallow depression "X" in the lateral wall of the tiny embryo's brain, there would be no optic groove, no optic vesicle, no optic cup, no eyeball, and no muscles for opening or closing the eyelids. It can be shown that most adult structures arise from the attempts of one part of the conceptus to counteract or balance the otherwise disruptive influences from another part: hence the reversal of many functions between embryonic stages and adulthood.

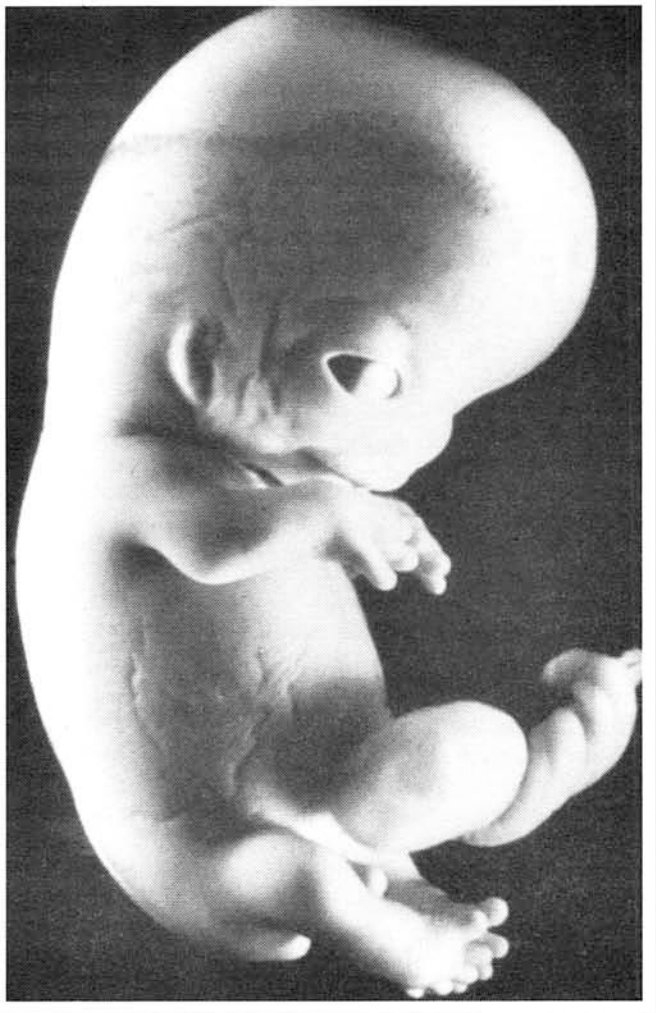

Fig. 7: Human embryo 23.6 mm long, 7–8 weeks after conception. Note the prominent forehead and the depression below it at the bridge of nose; also sharp border of upper and lower eyelids (distinct palpebral fissure).

CONNECTIONS OF THE EYE

Since the optic cup is an extension of the brain, the optic nerve too, is part of the brain, i.e., it is really a tract of the central nervous system rather than a peripheral nerve. Therefore changes inside the skull (such as elevated intracranial pressure) or within the eye socket (orbit), may cause changes along the optic nerve that manifest themselves at the site where the optic nerve leaves the eyeball. For example, the presence of a tumour growing inside the skull and causing an increased amount of fluid to form at the optic nerve head (papilloedema), might be revealed using an

Fig. 8: Fetus 50 mm long, about 9 weeks after conception. Note marked bridge of nose, fused eyelids and distinct margin of orbit.

ophthalmoscope, the instrument invented by Helmholtz in the 1850s to examine the fundus.

Most nerve fibres from the eye relay to the visual part of the cerebral cortex via the thalamus (lateral geniculate nucleus). The visual cortex, located near the occiput of the head, receives its oxygen and nutrients via the posterior cerebral artery, which is a continuation of the vertebral artery. On each side, the vertebral artery ascends to the brain through a series of lateral holes (transverse foramina) in the upper six vertebrae of the neck. Thus changes in the alignment of neck vertebrae (through accidents, or manipulation of the neck) can alter the blood supply to the visual cortex. Similarly, tension in muscles attached to the vertebrae of the neck may lead to changes in visual capacity. Furthermore, some optic fibres relay to a part of the brainstem called the superior colliculus, which has important connections to other parts of the brainstem that control eye movements, the position and thickness of the lens, and the diameter of the pupil. Most of these structures are also supplied by branches of the vertebral artery, so it is not surprising that the use of the neck, in F.M. Alexander's sense, may have widespread consequences for many aspects of vision, including the ability to focus the eye, or to follow moving objects.

THE VISUAL FIELD

The two optic nerves meet at the optic chiasm where more than half the nerve fibres from one eye cross to the opposite side of the brain, in such a way that each half of the primary visual cortex receives information from the opposite half of the visual world (visual hemifield). Much is written about how the cerebral cortex might resynthesise a single visual world (i.e., as seen by the "mind's eye") from these two hemifields. Here it is useful to remind ourselves that the retinas are also part of the brain and that each retina is "seeing" almost the same visual world that the mind "sees". ■

ENDNOTES

1. The refractive index is a measure of the capacity of a medium to bend a light ray, and is the ratio of the speed of light in air to its speed in the medium.
2. Where possible, technical terms are given in parentheses following the everyday term.
3. Teleological ideas still pervade contemporary developmental biology and "functional anatomy".
4. Lengths of embryos are measured from crown to rump.
5. The definition of the earliest precursor of any organ is somewhat arbitrary, since it is obviously a consequence of the development of earlier structures. Tracing any sequence backwards, one soon arrives at the single fertilised egg (conceptus) from which arises the whole embryo and all its surrounding fluids and membranes.
6. A process is a fibre-like prolongation or extension of the body of a cell; most nerve and glial cells in the brain have more than one process.
7. References to relevant scientific papers are available by contacting the author as below.

ACKNOWLEDGMENTS

The photographs were taken by the late Emeritus Professor Erich Blechschmidt. Sketches for figures 2-4 are based on material in the Blechschmidt Museum Collection of Human Embryos, University of Göttingen, Germany.

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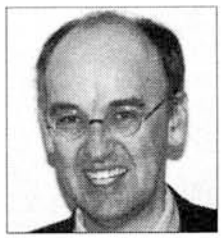
ABOUT THE WRITER



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