# Role of vision on early motor development: lessons from the blind

Heinz FR Prechtl\* DM DPhil FRCOG (hon), Professor Emeritus of Developmental Neurology, Department of Physiology, Karl Franzens University of Graz; Giovanni Cioni MD, Professor of Child Neurology and Psychiatry, INPE, University of Pisa and Stella Maris Scientific Institute, Pisa, Italy;

**Christa Einspieler** PhD, Associate Professor of Physiology, Department of Physiology, Karl Franzens University of Graz, Austria;

Arend F Bos MD PhD; Department of Paediatrics, Division of Neonatology, Beatrix Children's Hospital, University Hospital, Gröningen, The Netherlands;
Fabrizio Ferrari MD, Associate Professor of Neonatology, Department of Obstetrics and Paediatrics, University Hospital, Modena, Italy.

\**Correspondence to first author at* Department of Physiology, Karl Franzens University of Graz, Harrachgasse 21/5, A-8010 Graz, Austria. E-mail: christa.einspieler@kfunigraz.ac.at

For a better understanding of the contribution vision makes to the development of other sensory systems and to movement and posture, we studied effects of early blindness by examining video recordings of 14 totally blind infants. Infants were born at term or preterm and showed no evidence of brain damage. During preterm and term periods no noticeable changes in motor activity were observed. Around 2 months postterm all infants showed clear delay in head control and abnormal, exaggerated type of 'fidgety movements'. Later, postural control was characterized by a prolonged period of ataxic features. Results indicate a lack of normal calibration exerted by vision on proprioceptive and vestibular systems. Early visuomotor coordination such as coordinated eye—head scanning and head orientating were present but disappeared after several weeks.

The problem of early blindness is not restricted to the effects incurred by lack of vision. It also entails lack of calibration of other sensory systems exerted by vision. The vestibular system is affected. The proprioceptive system and development of cerebellar functions also seem to be impaired. Blind infants provide experimental evidence concerning the essential role of visual information in early motor development and how and when the absence of vision may be compensated for. It has long been known that the development of blind infants is delayed in various domains, especially in self-initiated postures and locomotion (Fraiberg 1977, Sonksen 1993, Tröster et al. 1994, Tobin et al. 1997). However, the problem with many studies on early blindness is the inclusion of infants who have additional brain damage. This makes it very difficult to distinguish beyond doubt which effects are due to lack of vision and which are functional sequelae of the infants' additional brain damage.

To explore this problem, we analysed lengthy and repeated video recordings of 14 carefully selected infants who were totally blind but without signs of brain damage. The other new aspect was that we were able to observe blind infants during their preterm and early postterm period.

## Method

From 1985 to 1995, 14 infants were recruited from departments of neonatology and ophthalmology in Pisa, Italy; Graz, Austria; and Gröningen, The Netherlands. They were selected on the following two criteria: (1) severe blindness checked by ophthalmological examination, and (2) no evidence of brain damage based on brain imaging and repeated neurological examinations. Absence of significant signs of brain damage was checked by weekly ultrasound examination during the preterm and early postterm period. During the second half of the first year MRI or CT were performed. Repeated careful neurological assessments employing methods described by Dubowitz and Dubowitz (1981) for preterm infants, by Prechtl (1977) for term infants, and Amiel-Tison and Grenier (1986) and Touwen (1976) for the first year of life, were carried out by our experts in the field. All observations of the infants' behaviour were based on video recordings. Since the video recordings were made for clinical case documentation, not all infants were recorded at exactly the same ages. We devoted careful attention to the qualitative aspects of various motor patterns. Of great importance to us was the age at which certain motor patterns appeared and disappeared and how they were performed. Of special interest were those aspects of motor patterns that blind infants may share with normally sighted infants, despite the fact that these motor patterns are generally considered to be visually elicited and guided. Figure 1 shows an analysis from video recordings of a blind infant at about 3 months postterm.

Cases of early blindness without detectable brain impairment are very rare (Jacobson et al. 1998). Nevertheless, we were able to recruit 14 infants (13 born preterm and one at term). Their main clinical data are shown in Table I. Some infants had mild and transient either periventricular increased echodensity at the brain ultrasound, or hypotonia, or hyperexcitability at the neurological examination performed at term age. At the last check they all were neurologically normal, but severely blind. They often showed some light perception, and three infants had some residual pattern vision at the lowest measurable spatial frequency. In the 13 preterm infants the cause of blindness was retinopathy of prematurity (ROP) grade 4 or 5 (Committee for the Classification of Retinopathy of Prematurity 1984, 1987) and in the infant born at term it was microphthalmia and cataract. All infants had been enrolled in a vigorous rehabilitation programme but symptoms persisted despite this careful intervention.

Thirteen infants were repeatedly video recorded during their first year of life (postterm age) and five also during their preterm period. One infant was only video recorded during the preterm period. We also have video recordings from the second and third year of life of four of the infants. This odd selection of observations was a matter of supply and not of study design. The limitations in recruiting adequate participants were formidable. We did not recruit a special control group for this study as we took normative data from previous studies, particularly on spontaneous movements and postures at early infancy (Cioni et al. 1989, Cioni and Prechtl 1990).

#### Results

During the preterm and term period, no single conspicuous behaviour pattern was observed which could indicate the infants' blindness. The repertoire of spontaneous motility appeared to be normal. As in sighted infants, the early-blind infant showed complex, fluent, and frequently occurring general movements (Prechtl 1990) i.e. movements involving all body parts. No differences were observed for isolated arm and leg movements, stretches, yawns, trunk and head rotations, nor for short phasic movements such as startles and twitches (Cioni and Prechtl 1990).

The first signs of developmental delay in relation to head control could be seen at the beginning of the 3rd month of postterm age when head lift in prone position was poor or absent. During the pull from supine into sitting position (traction test) all infants showed an abnormal head lag which continued until about 6 to 7 months' postterm age. Due to

the vestibular control, a normally developing infant keeps its head in the horizontal plane when the examiner tilts the infant sidewards, forwards, or backwards from an upright suspended body position. This response was absent in all infants at least until the end of the first year. This insensitivity suggests a delay in vestibular function due to the lack of visual calibration of the labyrinthine functions. In contrast to this delay we observed that, as in sighted infants (Cioni and Prechtl 1990), the infant's head was centred in midline at 8 to 10 weeks when lying in supine position. It is therefore most likely that the midline position of the head is not due to the vestibular but to sensory control by neck receptors. The prominent role of vestibular calibration is also supported by the observation that at the age of unsupported sitting and standing, (which are both grossly delayed) all infants kept their heads bent forward at an angle of about 30 degrees. The most likely explanation for this is that the horizontal semicircular canal is brought into its most sensitive position, namely horizontal orientation. This was seen particularly when the infant's posture was not supported. Infants' heads were raised to normal position if their trunks were supported or if they were listening to an interesting sound with focused attention.

A very striking feature concerned a peculiar type of 'fidgety movements'. All normally developing infants have a spontaneous movement pattern at postterm age of 9 to 15 weeks during wakefulness in which they move their arms and legs with graceful, small movements; trunk and neck muscles are also involved in this restlessness. We call this pattern 'fidgety movements' (Hopkins and Prechtl 1984). It should be mentioned that infants who do not develop fidgety movements are at a very high risk of developing major neurological deficits (Prechtl et al. 1997). In all our blind infants, observed during the relevant age range, fidgety movements were grossly disturbed in a specific way. They

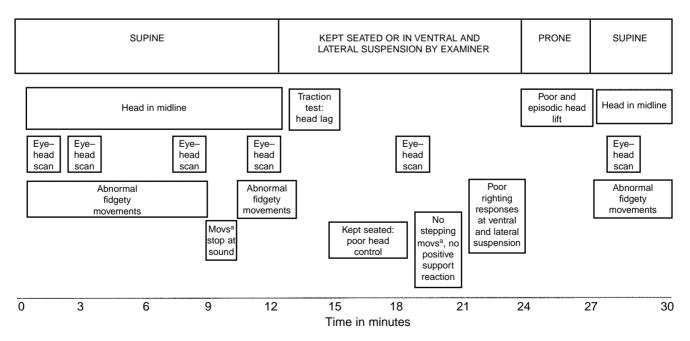


Figure 1: Actogram from video recording of a blind infant aged 11 weeks' postterm, manipulated for assessment of functions for part of time. <sup>a</sup>Movs, movements by infant.

were exaggerated in amplitude and jerky in character and their presence lasted longer than in sighted infants: until 8 to 10 months postterm age. Moreover, these movements were distinctly different from the abnormal fidgety movements seen in some infants with brain damage (Prechtl et al. 1997). In order to investigate if actual visual control is necessary for normal fidgety movements, six 3-month-old sighted, awake, infants were filmed in the dark with a special light-sensitive camera. Their fidgety movements did not change in character and continued to look normal. Experiments to blindfold these infants failed because they immediately protested by crying, which itself inhibits fidgety movements. We conjectured that the period of normal fidgety movements is necessary to calibrate the proprioceptive system (Prechtl et al. 1997). The observation of the early-blind infants supports this hypothesis. We speculate that exaggerated fidgety movements may indicate an attempt to compensate for the lack of integration of proprioception and vision.

Fine manipulation of objects is affected in blind infants as there is no visual monitoring of these voluntary movements. However, when fine manipulations in sighted infants, who do not actually look at their hands, are compared with those of blind infants of the same age, the latter are much more immature and clumsy. This again could be explained in terms of a delay in the development of the proprioceptive system, which lacks integration with vision. We do not yet know if catch-up occurs later or if proprioception remains insufficient.

Normally developing infants search and scan their environment in a consistent manner when they reach the age of 3 months. It was surprising to observe prolonged periods of scanning eye movements correlated with small scanning head movements in the blind infants, identical to those in sighted infants. The characteristic roving eye movements of blind people had developed in only three infants at this early age. Despite this fact, small saccadic head movements followed the same direction as the abnormal roving eye movements. However, this coordination disappeared after several weeks. When the blind infants could sit up with support at 6 to 9 months and an object such as a small cube was placed in one hand held in a lateral position, they immediately oriented their head by turning in the direction of the stimulated hand to 'look' at it. These events disappeared between the age of 10 to 18 months. This orienting response, it would seem, is built in and not generated by the visual input. As in the eye–head scanning movements, maintenance is normally provided by the visual system as this orienting response also disappeared in the blind but stayed on in the sighted child.

All infants in this study showed the expected delay in gaining postural control. This was not restricted to poor head control in the prone position (head lift), during the traction test, when sitting up or during unsupported sitting and standing. In addition, in these blind infants without brain damage we found long-lasting ataxic instability when they were brought to a sitting position or during free sitting. These ataxic movements included trunk and head and they often lasted until the child was 12 to 14 months old and then disappeared. Due to visual projection to the cerebellar vermis and cortex (Stein and Glickstein 1992) it could be assumed that a lack of this input leads to a delay in cerebellar control of balance in the sitting position and hence leads indirectly to a very prolonged period of postural instability, expressed as ataxia.

### Discussion

There are two possible reasons for the absence of noticeable effects of early blindness on spontaneous motor activity during the early weeks of life. First, it is uncertain if ROP affects

Patient nr	Gestational age (wk)	Birthweight (g)	Neuroimaging findings	Neurological examination at term	Ophthalmological findings	Duration of follow-up (mo)	Visual outcome	Neurological outcome
8	24	510	US: T-PVD MRI: N	Mild HPX	ROP 4	48	Totally blind	N
1	26	940	US: T-PVD MRI: N	Ν	ROP 5	24	Totally blind	Ν
4	26	800	US: T-PVD	Ν	ROP 4	28	Some residual	Ν
9	27	950	US: T-PVD	Mild HPO	ROP 5	72	Totally blind	Ν
14	27	970	US: T-PVD	Mild HPO	ROP 5	36	Totally blind	Ν
6	28	1280	US: N CT: N	Mild HPO	ROP 5	36	Totally blind	Ν
12	28	1180	US: N	Ν	ROP 5	24	Totally blind	Ν
2	29	1040	US: T-PVD MRI: N	Mild HPO	ROP 4	48	Some residual	Ν
7	29	1250	US: T-PVD	Ν	ROP 5	18	Some residual	Ν
13	29	1080	US: T-PVD	Ν	ROP 5	50	Totally blind	Ν
10	30	1460	US: T-PVD	Mild HPO	ROP 5	48	Totally blind	Ν
11	30	1350	US: N	Ν	ROP 4	48	Some residual	Ν
3	32	1150	US: N	Ν	ROP 5	36	Totally blind	Ν
5	40	2600	US: N MRI: N	Ν	Bi-micph and cataract	50	Totally blind	Ν

Table I: Main clinical data of 14 blind infants

US, brain ultrasound; T-PVD, transient (<15 days) periventricular increased echodensity; HPX, hyperexcitability; ROP, retinopathy of prematurity; N, normal; HPO, hypotonia; Bi-micph, bilateral microphthalmia.

vision during the first weeks of life before the detachment of the retina has occurred. More likely is the limited role of vision at this early age. During the first weeks after birth, van der Meer and coworkers (1995) found no effect of vision on spontaneous arm movements unless the weight of the moving arm was increased, which is an unnatural condition. It is not surprising therefore, to find no effect of blindness until the 3rd month postterm. At this age a major transformation of neural function occurs (Prechtl 1984) and many neural functions change into a more adaptive condition than during the first 2 months after birth at term (Prechtl 1986). The importance of vision for the development of vestibular control of posture has previously been shown (Matiello and Woollacott 1997). The same holds true for proprioception (Tröster et al. 1994). Our observations fully confirm these views in infants without brain damage. Thus, the delays mentioned are due to lack of vision and are not a consequence of additional brain dysfunction which could have affected the two sensory systems.

To find scanning movements and head orientating to tactile stimulation is in accordance with many other anticipatory movement patterns which are innate and not learned. They anticipate later functions similar to many foetal motor patterns, e.g. foetal eye movements or breathing movements (Prechtl 1989) or to smiling before 6 weeks postterm when social smiling starts to occur. Such built-in motor patterns have been extensively described in the ethological literature (e.g. Fentress 1992). It is important to mention that the maintenance of these motor patterns depends on the normal functioning of specific sensory systems.

#### Conclusion

During motor development, vision provides important feedback to the vestibular and proprioceptive systems; consequently, motor development is impeded in cases of early blindness. Our results provide evidence that from the first months onwards blindness does indeed affect early motor development. Strategies to compensate for the lack of calibration of the vestibular and proprioceptive systems provided by vision, should be improved in early therapeutic interventions for blind infants. It is an established fact that sensory systems have a wide range of plasticity and, to a certain degree, early intervention may help to compensate for the lack of vision.

#### Accepted for publication 8th June 2000.

#### Acknowledgements

We greatly acknowledge the cooperation of the institution 'Vision' in Graz, Dr A Boldrini (Pisa) and Dr G Rapisardi (Florence) for recruiting some of the infants and Dr P Paolicelli (Pisa) for some neurological examinations. Our gratitude goes to Dr T Brantsmavan Wulften Palthe in Utrecht, for her help in correcting the English. Our special thanks go to Professor Kevin Connolly (Sheffield) for his suggestions and advice.

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