



REVIEW ARTICLE

Vestibular Examination of Children With Alterations in Balance (I): Clinical and Instrumental Examination Methods^{☆,☆☆}

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Received 8 October 2010; accepted 3 January 2011

KEYWORDS

Dizziness;
Vestibulo-ocular
reflex;
Positional vertigo

PALABRAS CLAVE

Vértigo;
Mareo;
Inestabilidad;
Reflejo vestibulo-
oculomotor;
Postura Vertigo

Abstract This is a review of the normal methods for vestibular examination, both clinical and instrumental, to evaluate the vestibular system. Emphasis is given to the examination sequence and objectives in each method.

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Exploración vestibular de niños con alteraciones del equilibrio (I): métodos de la exploración clínica e instrumental

Resumen Se realiza una revisión de los métodos de la exploración vestibular oculomotora, postural y de las habilidades motrices necesarias para el equilibrio. Se hace hincapié en la secuencia de exploración y objetivos de cada método.

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Introduction

Balance alterations in children are not comparable to those of adults mainly because the aetiology is different, with

a predominance of benign paroxysmal positional vertigo (BPPV) of childhood and instability associated with otitis media with effusion. Neither must we forget that the response to vestibular damage through habituation and compensation is more efficient in children. Despite this, the initial clinical approach (anamnesis and clinical examination) is just as relevant as in adults and must be carried out in a systematic and structured manner.^{1,2} This problem cannot be separated from others that the child may suffer in everyday life (difficulties in learning, concentration, behaviour) or from the impact that a vestibular deficit may have on such important skills as visual acuity and reading ability.³ Consequently, we must work together with other specialists to achieve accurate, complete diagnosis, attempting to

[☆] Please cite this article as: Femia P, et al. Exploración vestibular de niños con alteraciones del equilibrio (I): métodos de la exploración clínica e instrumental. Acta Otorrinolaringol Esp. 2011;62:311–7.

^{☆☆} This work was prepared for the Congress of the European Society of Pediatric Otolaryngology held in 2010 in Pamplona, Spain.

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combine the four fundamental phenomena in this problem, the perceptual (vertigo), oculomotor (nystagmus), postural (instability) and vegetative (nausea and vomiting) with which to build a pathophysiological approach.

During the clinical examination, it is necessary to bear in mind that the results should be considered normal according to: (1) the child's age, (2) the maturation of vestibulo-ocular reflexes (VOR) and vestibulo-spinal reflexes (VSR), and (3) postural control. In general, we can say that the vestibular system is fully active at birth, but the saccadic system matures gradually until the age of 2 years.⁴ Therefore, fixing upon an object that appears in the visual periphery requires 2–3 saccades. The following system is ineffective at the beginning due to immaturity of foveal vision and lack of myelination, which is not complete until 5 months of age. Optokinetic nystagmus (OKN) is thus present from 6 months of age and already contains a slow following phase in the direction of the stimulus and a rapid phase (nystagmus) in the opposite direction; before 6 months, there is a tonic eye deviation (slow phase, first phase) without nystagmus. The VOR follows the same maturation programme as OKN but is subject to the controlling action of the visual system (visual-vestibular interaction), which will not be accurate and precise until the age of 10 months.

Ocular Motility and Spontaneous Nystagmus

The first few questions we ask about children are about their ocular motility: Do they move their eyes correctly? Is there nystagmus in primary or extreme positions of the gaze? Does visual fixation modify the above findings? If there is nystagmus, is it present from birth?

However, before answering these questions, if the child is helpful, a simple assessment of visual acuity can be made using a classic or modified Snellen chart with objects of interest to the child. In addition to giving information, the chart will make it possible to introduce the child to the exploration process in an entertaining, non-aggressive manner. Next, the head will be moved left and right, while requesting the child to continue reading or describing the figures; in this way, it is possible to measure "dynamic" visual acuity. This will be normal if the child keeps the same acuity as at rest or loses 1 or 2 lines, whereas it will be abnormal if, during mobilisation, the child needs large optotypes (more than 3 lines) to maintain visual acuity.⁵ When dynamic visual acuity is abnormal, a bilateral vestibulopathy should be suspected, requiring an oculomotor study and a basic vestibular reflex study.

The next step will be a complete exploration of the extrinsic ocular motility. This is controlled by pairs of muscles (straight and oblique) that cause eye movements on a voluntary basis and whose aim is to place the image of the object of interest in the area of greatest visual acuity, that is, the fovea. Saccades, following and fixing, will be studied. Saccades are studied between 2 points separated by a distance that allows the child to sweep an angle of 15–20°, observing the movement of both eyes; this study should not be too long, otherwise hypometria from inattention will quickly appear. Following an object of interest is the normal continuation of the previous study and the child should be

helped to complete it by avoiding staring at the examiner. With the exception of age, the OKN can be explored with a tape showing objects of interest or a manual rotating drum. Both in following and OKN, we should pay attention to the complete absence of ocular movement or to the more subtle and frequent asymmetric response. This part of the study ends by analysing whether nystagmus appears during ocular convergence.⁶

When nystagmus is observed while maintaining correct visual fixation, we should analyse whether it is a congenital nystagmus. This is present from birth, is usually pendular or even rotatory but without a defined rapid phase, does not alter visual acuity, disappears with convergence or when the direction of the gaze is somewhat deviated from the primary (which makes the child adopt a characteristic head position when attempting to minimise it); when looking upwards, it changes and becomes purely vestibular (with rapid and slow phases). Acquired nystagmus is vestibular and has a plane (horizontal, vertical or horizontal-rotary) and a direction (right, left, up, down, clockwise, counter-clockwise), making it possible to establish or initiate a differential diagnosis (Table 1). The situation changes when the nystagmus appears only when visual fixation is annulled: not only is it vestibular but is also possibly due to an alteration in the peripheral vestibular system, which includes the inner ear and the vestibular nerve.

Vestibulo-Oculomotor Reflex 1

How to know if the peripheral vestibular receptor is functioning normally? Exploration of VOR by clinical means is especially easy using the cephalic impulse or shake test described by Halmagyi and Gresty.⁷ A high-acceleration movement is applied to the head (low amplitude, high velocity) in one direction, while asking the child to look forward, fixing the stare on a close point. Under normal conditions, this causes a vestibular stimulus (amplifying or excitatory inertia-induced flow of the endolymph in the horizontal semicircular canal of the ear towards which the head moves) and a compensatory VOR moving the eye in the opposite direction. The end result is that the eye remains immobile in space, regardless of the displacement of the orbit. In case of a unilateral peripheral vestibular lesion, the reflex is incomplete or not proportionate to the degree of head displacement, so it is necessary to generate one or more saccadic movements in the direction opposite to the head movement to keep the eyes fixed on the desired point. This test can be performed on children from the age of 12 months and its results are sufficiently reproducible to provide highly useful information.⁸ Unfortunately, it is not easy to know the degree of canalicular paresis that represents an abnormal result, although in comparison with adults it may indicate a paresis of at least 40%.⁹

Next, while sitting on a parent's lap, the child is moved from side to side in the consultation chair; during this examination, which is usually well tolerated, the child wears Frenzel goggles or a videonystagmography mask momentarily to observe per-rotatory nystagmus. It may be easier to observe when it stops in one cycle, before resuming the movement in the opposite direction, mimicking post-rotatory nystagmus. The result is abnormal when no

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