SECTION D

Positional and positioning vertigo
Positional or positioning vertigo and nystagmus syndromes can be attributed to either peripheral or central vestibular dysfunction (Table D.1). The term positional signifies that an altered otolith input, which results from a newly assumed head position, is the causative mechanism. "Positioning" refers to the change in head position itself (head movement relative to the gravity vector) as the causative mechanism. The most common form is benign paroxysmal positioning vertigo (Chap. 16). It is due to canalolithiasis in the posterior semicircular canal (p-BPPV) or less frequently in the horizontal (h-BPPV) or anterior (a-BPPV) semicircular canal. Positional vertigo is also a characteristic symptom of perilymph fistulas (Chap. 6), Meniere’s disease (Chap. 5), and vestibular atelectasis.

Other labyrinthine manifestations such as positional alcohol nystagmus (p. 286), positional nystagmus with macroglobulinaemia (p. 288) and “heavy water” or glycerol ingestion (p. 287) occur because of a specific gravity differential between the cupula and the endolymph (buoyancy mechanism, Chap. 17). Neurovascular cross-compression of the vestibular nerve is the causative factor for vestibular paroxysmia (“disabling positional vertigo”), an insufficiently described entity (Chap. 17). Head-position dependent eighth nerve compression with positional vertigo may also be due to a mass in the cerebellopontine angle (p. 124).

Central positional vertigo (Chap. 18) is induced either by head movements, which result in a transient ischaemia of the pontomedullary brainstem (transient vertebrobasilar ischaemic attacks), or more commonly in archicerebellar lesions by a change in head position relative to the gravitational vector. The latter can take at least three forms: positional downbeat nystagmus (nodulus), positional nystagmus without concurrent vertigo and paroxysmal positional vertigo with nystagmus and/or nausea (vomiting). Central vestibular positional vertigo syndromes always indicate a dysfunction (disinhibition of otolith-canal interaction) of the infratentorial connections between the vestibular nuclei and the intra-axial vestibulocerebellar, in particular dorsal vermis, structures.

**Table D.1. Positional or positioning vertigo and nystagmus**

<table>
<thead>
<tr>
<th>Central vestibular (vestibular nuclei, dorsal vermis, vestibulocerebellar pathways)</th>
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<tbody>
<tr>
<td>Positional downbeat nystagmus</td>
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<tr>
<td>Central positional nystagmus without major vertigo</td>
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<tr>
<td>Central paroxysmal positional vertigo with nystagmus and/or nausea</td>
</tr>
<tr>
<td>Transient, head-position dependent vertebrobasilar ischaemia</td>
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</tbody>
</table>

**Vestibular nerve**

- Neurovascular cross-compression ("disabling positional vertigo": "vestibular paroxysmia")
- Head-position dependent eighth nerve compression with transient vestibular loss

**Peripheral labyrinth**

- Benign paroxysmal positioning vertigo (BPPV)
  - Of the posterior semicircular canal (p-BPPV)
  - Of the horizontal semicircular canal (h-BPPV)
  - Of the anterior semicircular canal (a-BPPV)
- Cupula/endolymph specific-gravity differential (buoyancy mechanism)
  - Positional alcohol vertigo/nystagmus (PAN)
  - Positional “heavy water” nystagmus
  - Positional glycerol nystagmus
  - Positional nystagmus with macroglobulinaemia
- Perilymph fistula
- Meniere’s disease
- Vestibular atelectasis

**Physiological vertigo**

- “Head (neck) extension vertigo”
- “Bending-over vertigo”

**Psychogenic positional/positioning vertigo**