Neurology of Vestibular Compensation

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Pathways to recovery from peripheral vestibular lesions

- Restoration of peripheral function
- Compensatory adjustments in brainstem circuits
- Substitution and Modulation
- Behavioral Changes
Vestibular regeneration doesn’t happen

Neurons don’t regenerate

In mammals, hair cells don’t regenerate
<table>
<thead>
<tr>
<th>Condition</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibular Neuritis</td>
<td>50% recover (2 months)</td>
</tr>
<tr>
<td>Bilateral Vest. Loss</td>
<td>Usually little (6 months)</td>
</tr>
<tr>
<td>BPPV</td>
<td>80% by 6 mo</td>
</tr>
<tr>
<td>Acoustic Neuroma</td>
<td>None</td>
</tr>
</tbody>
</table>
Two types of deficits after unilateral vestibular loss

- Static Imbalance
- Dynamic Imbalance

  - Spontaneous nystagmus
  - Movement induced visual disturbance
Responses at brainstem level to unilateral loss of vestibular function: 

**Static Imbalance**

- Tone asymmetry at VN level including some silent neurons on deficit side. **Tone is equalized rapidly in days-week** (Markham, 1984)

Compensation occurs rapidly
Does not require vision or movement
Responses at brainstem level to unilateral loss of vestibular function

**Dynamic Compensation**

- Reduced dynamic sensitivity because of loss of 50% of peripheral input. Gradual increase in sensitivity over months.
Cerebellar Clamp


Clamp may account for poorer adaptation in animals with lesions than normal persons with magnifying glasses.
Compensation for Dynamic Imbalance

- Occurs slowly (month or more)
- Requires sensory input
- Not robust -- lost after occipital lobectomy (Fetter and Zee, 1988) or lesions to vestibulocerebellum (Robinson, 1976)
What do we need to recover?

<table>
<thead>
<tr>
<th>Condition</th>
<th>Lesion</th>
<th>Need</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vestibular Neuritis</td>
<td>50%</td>
<td>1-2x</td>
</tr>
<tr>
<td>Acoustic Neuroma</td>
<td>50%</td>
<td>2x</td>
</tr>
<tr>
<td>Bilateral</td>
<td>50-100%</td>
<td>2x-infinite</td>
</tr>
<tr>
<td>BPPV</td>
<td>1 canal</td>
<td>suppress 1 canal</td>
</tr>
</tbody>
</table>
Factors Influencing Recovery

- **Promote Recovery**
  - Extensive Lesion
  - Ganglion preserved
  - Youth
  - Intact cerebellum
  - Intact senses
  - High physical activity

- **Reduce Recovery**
  - Minor lesion
  - Ganglion gone
  - Age
  - Damaged cerebellum
  - Diminished senses
  - Low general activity
Central Readjustments: How much improvement in gain is possible?

<table>
<thead>
<tr>
<th>Need</th>
<th>Response</th>
<th>Species</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>2x (spectacle)</td>
<td>1.7-1.9</td>
<td>Monkey</td>
<td>Miles/Eighmy</td>
</tr>
<tr>
<td>2x (canal plug)</td>
<td>1.6</td>
<td>Monkey</td>
<td>Paige, 1983</td>
</tr>
<tr>
<td>2x (labyrinthect)</td>
<td>1.5x</td>
<td>Cat</td>
<td>Cass and Goshgarian</td>
</tr>
</tbody>
</table>
## Critical Period

<table>
<thead>
<tr>
<th>Response</th>
<th>Present?</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postural Control</td>
<td>Yes</td>
<td>Lacour et al, 1989 (Baboon)</td>
</tr>
<tr>
<td>Postural Control</td>
<td>Yes</td>
<td>Jensen, 1979 (Guinea pig)</td>
</tr>
<tr>
<td>VOR-low velocity</td>
<td>No</td>
<td>Fetter and Zee, 1988 (Monkey)</td>
</tr>
<tr>
<td>VOR - high vel.</td>
<td>Yes</td>
<td>Fetter and Zee, 1988</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>No</td>
<td>Lacour et al, 1976</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>No</td>
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</tbody>
</table>
Context Specificity

VOR gain adaptation can be trained to be dependent on orientation to gravity as well as can be tuned to particular frequencies (species: Cat)

Powell et al (1991)
Substitution and Modulation Mechanisms in “parallel” with lower level brainstem reajustments

- Vision
- Other vestibular inputs (e.g. otoliths)
- Somatosensory (e.g. COR)
- Prediction
- Suppression of oscillopsia (cognitive)
- Modulation of VOR or COR gain
Use of Prediction

G. S. (Bilateral Vestibular loss from Gentamicin)

“I’m doing better but I still have trouble when I pivot or turn in an unplanned manner”
Behavioral Changes (cognitive)

- Limit head movement
- Limit body movement
- Take more precautions
Drugs that Accelerate Dynamic Compensation (in animals)

- Amphetamines (1986)
- Bromocriptine (Dopamine agonist)
- ACTH (adreno-cortico-trophic hormone)
- Caffeine
- TRH
- Antiemetic given transiently (Peppard, 1986)

Modified from Brandt, 1991, also Peppard (1986)
Drugs that Retard Dynamic Compensation in animals

- Phenobarbital (sedative, Barbituate)
- Lisuride (Dopamine antagonist)
- ACTH antagonists
- Chlorpromazine (Thorazine)
- Diazepam (Valium) – if long term only
- Dimenhydrinate (Peppard, 1986)
- Scopolamine (Peppard, 1986)

Modified from Brandt, 1991; Also Peppard, 1986
Drugs that have no effect on Compensation

• Baclofen

Summary

Recovery from Vestibular lesions occurs via multiple redundant mechanisms

- Peripheral recovery is variable
- Static compensation is rapid and robust
- Dynamic compensation is slow, fragile and incomplete
- In general, sedatives impair dynamic compensation and stimulants enhance compensation
Summary - II

- Dynamic compensation is unlikely to be adequate for lesions involving more than 50% of vestibular periphery.

- Substitution, Modulation, and Behavioral Changes provide other important routes for recovery.