Aangepaste richtlijn Benigne Paroxysmale Positieduizeligheid (BPPD)

INITIATIEF

Nederlandse Vereniging voor Keel-Neus-Oorheelkunde en Heelkunde van het Hoofd-Halsgebied

in samenwerking met de Nederlandse Vereniging voor Neurologie

MET ONDERSTEUNING VAN

Orde van Medisch Specialisten

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COLOFON

Richtlijn Adaptie BPPD

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De werkgroep werd methodologisch ondersteund door de afdeling Ondersteuning Professionele Kwaliteit van de Orde van Medisch Specialisten in de personen van ir. T.A. van Barneveld (afdelingshoofd) en Dr. M. Molag (adviseur).

De richtlijn betreft een adaptatie van:

Clinical practice guideline: Benigne Paroxysmale Positionele Duizeligheid.
De Amerikaanse richtlijn van de Academy of Otolaryngology-Head and Neck surgery foundation 'Clinical practice guideline: Benign paroxysmal positional vertigo' (Bhattacharayya, et al., 2008) vormde het uitgangspunt van de onderhavige richtlijn. Daarnaast werd de Amerikaanse richtlijn van de Academy of neurology, gericht op de behandeling van BPPD, gebruikt ter aanvulling (Fife, et al., 2008), alsmede de discussies en richtlijnen van de Standaardisatie commissie van de Barany Society (Reykjavik, et al., 2010, www.baranysociety.nl).

Samenvatting richtlijn BPPD

BPPD wordt in de anamnese herkend als kortdurende draaiduizeligheid die wordt uitgelokt door een standsverandering van het hoofd ten opzichte van de zwaartekrachtsvector. In de dagelijkse praktijk manifesteert zich een BPPD vooral bij omdraaien in bed , gaan liggen in bed en omhoog kijken.

Samenvatting van de aanbevelingen

Onderstaande is een samenvatting van de belangrijkste aanbevelingen uit de evidence-based klinische richtlijn 'Benigne Paroxysmale Positionele Duizeligheid (BPPD)'. De Amerikaanse richtlijn van de Academy of Otolaryngology-Head and Neck surgery foundation 'Clinical practice guideline: Benign paroxysmal positional vertigo' (Bhattacharayya, 2008) vormde het uitgangspunt van de onderhavige richtlijn. Daarnaast werd de Amerikaanse richtlijn van de Academy of neurology, gericht op de behandeling van BPPD, gebruikt ter aanvulling (Fife, 2008), alsmede de discussies en richtlijnen van de Standaardisatie commissie van de Barany Society (Reykjavik, 2010, www.baranysociety.nl). Onze doelstelling was om deze multidisciplinaire richtlijn aan te passen aan de Nederlandse situatie met behulp van Nederlandse input, waarbij de aanbevelingen rekening houden met wetenschappelijk bewijs en zich richten op harm-benefit balans, en expert consensus om de gaten in wetenschappelijk bewijs op te vullen.
Primaire doelstellingen van deze richtlijn zijn: De kwaliteit van de zorg te verbeteren door middel van een accurate en snelle diagnose van BPPD; Voorkomen van onnodig gebruik van medicijnen; Doelgericht gebruik van aanvullend onderzoek; Stimuleren van het gebruik van repositiemanoeuvres als therapie voor BPPD.
In deze samenvatting ontbreken het wetenschappelijk bewijs en de overwegingen die tot de aanbevelingen geleid hebben. Lezers van deze samenvatting worden voor deze informatie verwezen naar de volledige richtlijntekst. Deze samenvatting van aanbevelingen staat niet op zichzelf. Bij medische besluitvorming dient rekening te worden gehouden met de omstandigheden en voorkeuren van de patiënt. Behandeling en procedures met betrekking tot de individuele patiënt berusten op wederzijdse communicatie tussen patiënt, arts en andere zorgverleners.

Diagnostiek BPPD

<u>Wat is de beste manier om BPPD van het posterieure kanaal (p-BPPD) te diagnosticeren?</u>

- 1. De diagnose p-BPPD (zekere BPPD) wordt gesteld wanneer draaiduizeligheid met upbeat, rotatoire nystagmus wordt opgewekt door de Dix-Hallpike manoeuvre.

<u>Wat is de beste manier om BPPD van het horizontale kanaal **(h-BPPD)** te diagnosticeren?</u>

- De diagnose h-BPPD wordt gesteld wanneer draaiduizeligheid met horizontale nystagmus wordt opgewekt door de supine roll test.
- Als de patiënt een anamnese heeft die past bij BPPD maar de Dix-Hallpike is negatief, moet een BPPD van het horizontale kanaal worden overwogen en een 'supine roll' test worden gedaan.

<u>Wat is de beste manier om BPPD van het anterieure kanaal **(a-BPPD)** te herkennen?</u>

De diagnose a-BPPD wordt gesteld wanneer draaiduizeligheid met downbeat nystagmus wordt opgewekt door de Dix-Hallpikemanoevre.

<u>Van welke andere vormen van positioneringsduizeligheid moet BPPD worden onderscheiden?</u>

- BPPD moet met name gedifferentieerd worden van andere aandoeningen die zich presenteren met houdingsafhankelijke duizeligheid, zoals bijvoorbeeld orthostatische hypotensie, vestibulaire uitval

Beeldvormend, audiologisch en vestibulair onderzoek

Wat zijn de indicaties voor beeldvormend, audiologisch en vestibulair onderzoek bij verdenking op BPPD?

- Beeldvormende technieken zijn niet geïndiceerd bij de diagnose BPPD. Beeldvormende technieken dienen wel te worden toegepast bij patiënten bij wie twijfel bestaat omtrent de diagnose BPPD, bij voorbeeld als additionele neurologische uitvalssymptomen aanwezig zijn, of bij therapieresistente BPPD.
- Vestibulaire functietesten hebben geen toegevoegde waarde bij patiënten met BPPD. Vestibulaire functietesten zijn alleen geïndiceerd bij patiënten met: 1) atypische nystagmus bij de diagnostische manoeuvres voor BPPD, 2) verdenking op additionele vestibulaire pathologie 3) een falende (of herhaaldelijk falende) reactie op canalith repositiemanoeuvres (CRM), of 4) frequent recidiverende BPPD.

Kunnen audiometrische testen de diagnose BPPD ondersteunen?

- Er is bij BPPD geen indicatie voor het uitvoeren van audiometrisch onderzoek.

Behandeling BPPD

Zijn repositiemanoeuvres geschikt als therapie om patiënten met BPPD te behandelen?

- Bij een zekere en waarschijnlijke p-BPPD is behandeling met een Epley of Semontmanoeuvre geïndiceerd.
- Er zijn aanwijzingen dat bij een h-BPPD behandeling met de log-roll manoeuvre of Lempertmanoeuvre effectief is.
- Er is onvoldoende bekend over de effectiviteit van repositiemanoeuvres bij de behandeling van a-BPPD.
- Behandeling van BPPD dient door een arts of specifiek daartoe geschoolde paramedicus te geschieden.

Is vestibulaire revalidatie geschikt als therapie om patiënten met BPPD te behandelen?

- Vestibulaire revalidatie is niet primair geïndiceerd bij BPPD.

Zijn medicijnen geschikt als therapie om patiënten met BPPD te behandelen?

- Er is geen indicatie om patiënten met BPPD medicatie voor te schrijven.

Is chirurgische interventie geschikt als behandeling?

- De werkgroep beveelt aan om bij ernstig invaliderende, therapieresistente, zekere BPPD canal plugging te overwegen. De kans op ernstige bijwerkingen zoals doofheid en blijvende evenwichtsstoornissen dient goed met de patiënt te worden besproken.
- Neurectomie van de n.singularis is een techniek die gezien de complexiteit voorbehouden is aan gespecialiseerde KNO-artsen.

Kan bij patiënten met BPPD volstaan worden met een expectatief beleid?

- Behandelen van BPPD verdient de voorkeur boven het afwachten van het natuurlijk beloop.

Met welke factoren moet rekening worden gehouden bij de behandeling van BPPD?

- Artsen dienen factoren die een verhoogd risico op vallen geven uit te vragen; dit beïnvloedt de behandelkeus van BPPD (voorkeur voor niet-conservatieve behandeling).

Is het noodzakelijk om de respons op BPPD behandeling te evalueren?

- Een maand na de behandeling dient het effect van de behandeling geëvalueerd te worden.

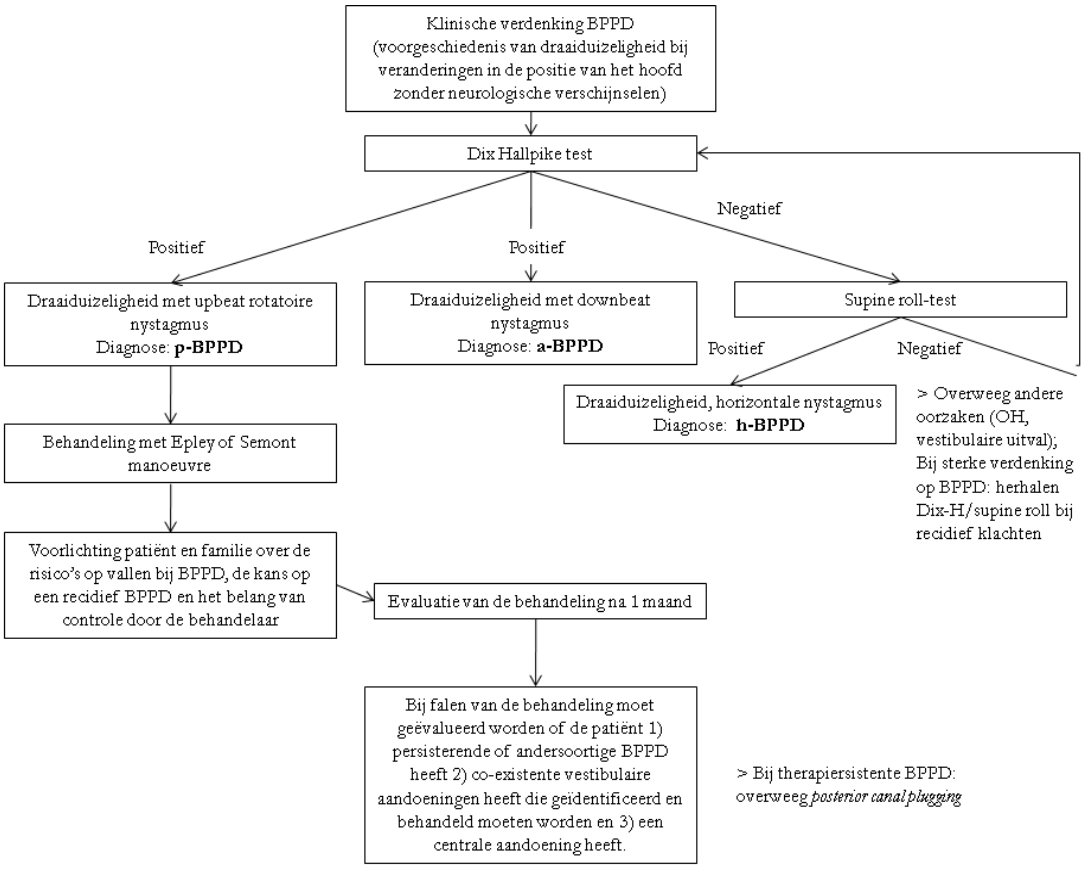
Hoe zou de evaluatie van de BPPD behandeling eruit moeten zien?

- Bij falen van de behandeling moet geëvalueerd worden of de patiënt 1) persisterende of andersoortige BPPD heeft 2) co-existente vestibulaire aandoeningen heeft die geïdentificeerd en behandeld moeten worden en 3) een centrale aandoening heeft.

Ten aanzien van welke aspecten rond BPPD zouden patiënten voorgelicht moeten worden?

- De patiënt dient verteld te worden dat BPPD een goedaardige, meestal goed behandelbare aandoening is. Het is belangrijk dat de patiënt (eventueel ook de familie) goed voorgelicht wordt over de risico's op vallen bij BPPD, de aanzienlijke kans op een recidief BPPD en het belang van controle door de behandelaar.

Stroomschema diagnose en behandeling van BPPD



Inhoudsopgave

Hoofdstuk 1 Algemene Inleiding

Achtergrond

Duizeligheid is een veel voorkomende aandoening. Op basis van een groot onderzoek in de algemene populatie werd vastgesteld dat de 1-jaars prevalentie voor duizeligheid 4.9% is en voor BPPD 1.6% (Neuhauser, et al., 2007). In de tweede lijn, in een gespecialiseerd duizeligheidscentrum, werd gevonden dat BPPD in 22,1% van de gevallen de oorzaak van de duizeligheid is (Bruintjes, et al., 2007). In de VS zou het percentage duizeligheid dat veroorzaakt wordt door BPPD zelfs 17-42% zijn (Schnappert, et al., 1992) (Katsarkas, et al., 1999) (Hanley, et al., 2001).

- Benigne paroxysmale positieduizeligheid (BPPD) wordt gedefinieerd als kortdurende draaiduizeligheid die wordt uitgelokt door een standsverandering van het hoofd ten opzichte van de zwaartekrachtsvector.
- Traditioneel gezien worden de termen benigne en paroxysmaal gebruikt om deze vorm van positionele vertigo te beschrijven. De term benigne geeft aan dat de positionele vertigo goedaardig is (niet veroorzaakt door een ernstige stoornis van het centraal zenuwstelsel) en dat de prognose meestal gunstig is (Baloh, et al., 2001).

Hoewel goedaardig, heeft niet gediagnosticeerde en onbehandelde BPPD een negatieve invloed op het functioneren (werkverzuim), de gezondheid en kwaliteit van leven (Lopez-Escamez et al., 2003; von Brevern et al., 2007). BPPD werd ook wel benigne postionele vertigo, paroxysmale positionele vertigo ofpositionele vertigo genoemd en direct gerelateerd aan benign paroxysmale nystagmus, en paroxysmale positionele nystagmus. In deze richtlijn koos de werkgroep ervoor om consequent de term benigne paroxysmale positieduizeligheid (BPPD) te hanteren, omdat dit de meest gebruikte terminologie is in de literatuur en de klinische praktijk.

BPPD komt meestal voor in een van de twee volgende varianten: BPPD van het posterieure semicirculaire kanaal (posterior canal BPPV) of BPPD van het horizontale semicirculaire kanaal (horizontal canal BPPV) (White, et al., 2005) (Cakir, et al., 2006) (Parnes, et al., 2003). Posterieur kanaal BPPD komt vaker voor dan horizontaal kanaal BPPD, p-BPPD is verantwoordelijk voor ongeveer 85 tot 95 procent van de gevallen van BPPD (Parnes, et al., 2003). De derde variant, de anterieur kanaal BPPD komt voor maar is zeldzaam (Korres, et al., 2002).

Hoewel onderwerp van discussie omdat er nog relatief weinig basaal wetenschappelijk bewijs is, wordt er wereldwijd van uitgegaan dat BPPD meestal wordt veroorzaakt door canalolithiasis en cupulolithiasis. Gruis (waarschijnlijk gefragmenteerde otoconiën die het kanaal binnenkomen) blijft respectievelijk steken en veroorzaakt veranderingen in de massatraagheid in het kanaal of blijft vastzitten op de cupula, resulterend in een nystagmus en vertigo bij standsveranderingen van het kanaal ten opzichte van de zwaartekracht (Parnes, et al., 2003) Parnes, et al., 1992). Fysische modellen (Rajguru, et al., 2004) en peroperatieve observaties (Parnes, et al., pc) ondersteunen de canalolitihiasis en cupulolithiasis theorien.

Horizontale kanaal BPPD veroorzaakt ongeveer 5 tot 15 procent van de gevallen van BPPD (White, et al., 2005) (Cakir, et al., 2006). De etiologie van horizontale kanaal BPPD lijkt ook te worden veroorzaakt door de aanwezigheid van gruis binnen het horizontale kanaal. De pathofysiologie wordt niet zo goed begrepen als die van posterieur kanaal BPPD. De andere zeldzame varianten zijn anterieur kanaal BPPD, multipele kanaal BPPD en dubbelzijdige kanaal BPPD en 'canalith jam' (kanaalverstopping). Een uitgebreide bespreking van deze zeldzame varianten valt buiten het bestek van deze richtlijn.

Het is nog onduidelijk wat de primaire oorzaak van het ontstaan van gruis in de kanalen of het ontstaan van neerslag op de cupula is. Veelvuldig genoemde mogelijke oorzaken zijn: hoofdtrauma (mechanische beschadiging van het statolieten membraan), lange bedrust, storingen in het Ca++ metabolisme, ziekte van het labyrinth (labyrinthitis, vasculaire problematiek enz).

Onderwerp en doel

De primaire doelstellingen van deze richtlijn zijn:

- De kwaliteit van de zorg te verbeteren door middel van een accurate en snelle diagnose van BPPD.
- Voorkomen van onnodig gebruik van medicijnen.
- Doelgericht gebruik van aanvullend onderzoek.
- Stimuleren van het gebruik van repositiemanoeuvres als therapie voor BPPD.

Secundaire doelstellingen zijn: beperking van de kosten van diagnose en behandeling van BPPD, vermindering van het aantal artsenbezoeken, en verbetering van de kwaliteit van leven. Het grote aantal patiënten met BPPD en de verscheidenheid aan diagnostische en therapeutische interventies voor BPPD maakt dit een geschikt onderwerp voor een evidence-based richtlijn.
De Amerikaanse richtlijn van de Academy of Otolaryngology-Head and Neck surgery foundation 'Clinical practice guideline: Benign paroxysmal positional vertigo' (Bhattacharayya, et al., 2008) vormde het uitgangspunt van de onderhavige richtlijn. Daarnaast werd de Amerikaanse richtlijn van de Academy of neurology, gericht op de behandeling van BPPD, gebruikt ter aanvulling (Fife, et al., 2008), alsmede de discussies en richtlijnen van de Standaardisatie commissie van de Barany Society (Reykjavik, et al., 2010, www.baranysociety.nl)). Onze doelstelling was om deze multidisciplinaire richtlijn te adapteren aan de Nederlandse situatie met behulp van Nederlandse input, waarbij de aanbevelingen rekening houden met wetenschappelijk bewijs en zich richten op harm-benefit balans, en expert consensus om de gaten in wetenschappelijk bewijs op te vullen. Deze specifieke aanbevelingen kunnen dan gebruikt worden om indicatoren te ontwikkelen en te gebruiken voor kwaliteitsverbetering.

Afbakening

Deze richtlijn heeft betrekking op patiënten met een klinische diagnose van BPPD, hierbij wordt opgemerkt dat er weinig tot geen literatuur beschikbaar is voor patiënten onder de 18 jaar. Deze richtlijn richt zich op BPPD, daarbij in aanmerking nemend dat BPPD kan ontstaan tegelijk met andere neurologische aandoeningen en dat de behandeling van de BPPD symptomen dan nog steeds volgens de richtlijn plaats hoort te vinden.

Richtlijngebruikers

Deze richtlijn is opgesteld voor KNO-artsen en neurologen die in hun klinische praktijk in aanraking komen met BPPD. De richtlijn is toepasbaar in iedere setting waar BPPD gediagnosticeerd en behandeld wordt.

Uitgangsvragen

In de richtlijn zullen de volgende uitgangsvragen worden beantwoord:

1. Wat is de beste manier om BPPD te diagnosticeren?
2. Van welke andere vormen van positioneringsduizeligheid moet BPPD worden onderscheiden?
3. Wat zijn de indicaties voor aanvullend onderzoek bij verdenking op BPPD?
4. Welke therapieën zijn geschikt om BPPD te behandelen?
    a. Zijn repositiemanoeuvres geschikt als therapie om patiënten met BPPD te behandelen?
    b. Is vestibulaire revalidatie geschikt om patiënten met BPPD te behandelen?
    c. Zijn medicijnen geschikt als therapie om patiënten met BPPD te behandelen?
    d. Is chirurgische interventie geschikt als behandeling?
    e. Kan bij patiënten met BPPD volstaan worden met een expectatief beleid?
5. Met welke factoren moet rekening gehouden worden bij de behandeling van BPPD?
6. Is het noodzakelijk om de respons op BPPD behandeling te evalueren en hoe zou deze evaluatie eruit moeten zien?
7. Ten aanzien van welke aspecten rond BPPD zouden patiënten voorgelicht moeten worden?

Samenstelling van de werkgroep

Deze werkgroep bestaat uit twee KNO-artsen, een neuroloog en een klinisch fysicus-vestibuloloog. Bij het samenstellen van de werkgroep is zoveel mogelijk rekening gehouden met specifieke deskundigheid en de geografische spreiding van de werkgroepleden en een evenredige vertegenwoordiging vanuit perifere en academische ziekenhuizen. De werkgroepleden hebben onafhankelijk gehandeld en waren vrij van financiële of zakelijke belangen betreffende het onderwerp van de richtlijn.

Werkwijze werkgroep

De Amerikaanse richtlijn van de Academy of Otolaryngology-Head and Neck surgery foundation ‘Clinical practice guideline: Benign paroxysmal positional vertigo’ (Bhattacharayya, et al., 2008) vormde het uitgangspunt van de onderhavige richtlijn. Daarnaast werd de Amerikaanse richtlijn van de Academy of neurology gebruikt (Fife, et al., 2008). Dit betekent dat de Nederlandse richtlijncommissie de studies, de beoordeling & gradering ervan en de begeleidende tekst heeft overgenomen. Studies, relevant voor dit onderwerp, die *nadien* werden gepubliceerd konden in de richtlijncommissie worden ingebracht. De literatuur werd bovendien geupdate door te zoeken in Medline naar nieuw verschenen systematische reviews en RCTs met als onderwerp BPPD in de periode van 2008 t/m 2010.

De richtlijncommissie is voor elke aanbeveling in de Amerikaanse richtlijn nagegaan welke overwegingen naast het wetenschappelijk bewijs zijn gebruikt en of de door de commissie aangedragen studies de aanbeveling zouden kunnen veranderen. Wanneer er consensus was over

deze overwegingen en door de commissie aangedragen studies geen ander inzicht opleverden, zijn de aanbevelingen overgenomen. Indien de commissie andere overwegingen (ook) van belang achtte of meende dat de door haar aangedragen studies een (iets) ander licht wierpen op de in de Amerikaanse richtlijn vermelde aanbeveling, zijn de aanbevelingen gemodificeerd.

De gradering van de studies in de Amerikaanse richtlijn wijkt af van wat hier te lande gangbaar is. Vanuit het oogpunt van uniformiteit achtte de Nederlandse commissie het wenselijk de classificatie van bewijs c.q. gradering te converteren naar de Nederlandse classificatie. De Amerikaanse classificatie is hieronder afgebeeld in tabel. De corresponderende “Nederlandse” classificatie is in tabel 1.2 opgenomen.

Tabel 1.1: gradering van de studies in de Amerikaanse richtlijn

Evidence quality for grades of evidence

Grade	Evidence quality
A	Well-designed randomized controlled trials or diagnostic studies performed on a population similar to the guideline’s target population
B	Randomized controlled trials or diagnostic studies with minor limitations; overwhelmingly consistent evidence from observational studies
C	Observational studies (case-control and cohort design)
D	Expert opinion, case reports, reasoning from first principles (bench research or animal studies)
X	Exceptional situations for which validating studies cannot be performed and there is a clear preponderance of benefit over harm

Tabel 1.2 Relatie tussen Evidence quality for grades of evidence en niveau van conclusie op basis van kwaliteit van bewijs conform Classificatieschema van CBO.

Evidence Quality - symbool	Evidence Quality – omschrijving	Niveau van conclusie – symbool	Niveau van conclusie omschrijving
A	Well-designed randomized controlled trials or diagnostic studies performed on a population similar to the guideline's target population	1	Meerdere gerandomiseerde dubbelblinde vergelijkende klinisch onderzoeken van goede kwaliteit van voldoende omvang, of Meerdere onderzoeken ten opzichte van een referentietest (een 'gouden standaard') met tevoren gedefinieerde afkapwaarden en onafhankelijke beoordeling van de resultaten van test en gouden standaard, betreffende een voldoende grote serie van opeenvolgende patiënten die allen de index- en referentietest hebben gehad
B	Randomized controlled trials or diagnostic studies with minor limitations; overwhelmingly consistent evidence from observational studies	2	Meerdere vergelijkende onderzoeken, maar niet met alle kenmerken als genoemd onder 1 (hieronder valt ook patiënt-controle onderzoek, cohort-onderzoek), of Meerdere onderzoeken ten opzichte van een referentietest, maar niet met alle kenmerken die onder 1 zijn genoemd.
C	Observational studies (case-control and cohort design)		
D	Expert opinion, case reports, reasoning from first principles (bench research or animal studies)	3 en 4	Niet vergelijkend onderzoek of mening van deskundigen

In de Amerikaanse richtlijn worden ook de aanbevelingen gegradeerd in termen van 'strong recommendation', 'recommendation', 'option'. Hier te lande is graderen van aanbevelingen niet gebruikelijk. Om deze reden zijn in de Nederlandse richtlijn de aanbevelingen niet gegradeerd.

De literatuurzoekstrategie die de Amerikaanse richtlijncommissie heeft gevolgd, staat in bijlage 1 beschreven. Voor het opstellen van de aanbevelingen heeft de Amerikaanse richtlijncommissie gebruik gemaakt van de *GuideLine Implementability Appraisal* (GLIA) *tool*. Dit instrument dient om de helderheid van de aanbevelingen te verbeteren en potentiële belemmeringen voor de implementatie te voorspellen. In bijlage 3 wordt een aantal criteria beschreven. Ook de Nederlandse richtlijncommissie heeft deze criteria gehanteerd

Juridische betekenis van richtlijnen

Richtlijnen zijn geen wettelijke voorschriften, maar bevatten expliciete, zo veel mogelijk op evidence gebaseerde aanbevelingen en inzichten waaraan zorgverleners zouden moeten voldoen om kwalitatief optimale zorg te verlenen. Aangezien deze aanbevelingen hoofdzakelijk gericht zijn op de 'gemiddelde patiënt', kunnen zorgverleners op basis van individuele patiëntkenmerken zo nodig afwijken van de richtlijn. Afwijken van richtlijnen is, als de situatie van de individuele patiënt dat vereist, soms zelfs noodzakelijk. Een richtlijn kan worden gezien als een papieren weergave van een best practice. Als van de richtlijn wordt afgeweken, is het raadzaam dit gedocumenteerd en beargumenteerd te doen.

Herziening

De richtlijn zal na 3 jaar worden getoetst aan de wetenschappelijke ontwikkelingen door een (nog samen te stellen) multidisciplinaire commissie. De commissie draagt de verantwoordelijkheid om tussentijdse peilingen bij de beroepsgroepen te verrichten naar behoefte voor herziening(en) van de huidige richtlijn. Bij essentiële ontwikkelingen kan er besloten worden tussentijdse elektronische amendementen te maken en deze onder de verschillende beroepsgroepen te verspreiden. Zo nodig wordt een nieuwe werkgroep geïnstalleerd om (delen van) de richtlijn te herzien. Uiterlijk in 2014 zal een nieuwe multidisciplinaire werkgroep worden geïnstalleerd voor een volledig herziene versie van de richtlijn.

Personele samenstelling werkgroep

- dr. Tj.D. Bruintjes (voorzitter), KNO-arts, Gelre ziekenhuizen, Apeldoorn
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- dr. ir. T van Barneveld klinisch epidemioloog, Orde van Medisch specialisten (adviseur)
- dr. M.L. Molag, Orde van Medisch specialisten (adviseur)

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Hoofdstuk 2 Diagnose BPPD

Uitgangsvraag 1a:

Wat is de beste manier om BPPD van het posterieure kanaal (p-BPPD) te diagnosticeren?

Posterior semicircular canal BPPV is diagnosed when 1) patients report a history of vertigo provoked by changes in head position relative to the gravity vector and 2) when, on physical examination, characteristic nystagmus is provoked by the Dix-Hallpike maneuver (Table 2.1).

Table 2.1. Diagnostic criteria for posterior canal BPPV

History	Patient reports repeated episodes of vertigo with changes in head position.
Physical examination	Each of the following criteria are fulfilled: ● Vertigo associated with nystagmus is provoked by the Dix-Hallpike test. ● There is a latency period between the completion of the Dix-Hallpike test and the onset of vertigo and nystagmus. ● The provoked vertigo and nystagmus increase and then resolve within a time period of 60 seconds from onset of nystagmus.

History

Vertigo has been defined as an “illusory sensation of motion of either the self or the surroundings.” (Blakley, et al., 2001) The symptoms of vertigo resulting from posterior canal BPPV are typically described by the patient as a rotational or spinning sensation when the patient changes head position relative to gravity.

The episodes are often provoked by everyday activities and commonly occur when rolling over in bed or when the patient is tilting the head to look upward (eg, to place an object on a shelf higher than the head) or bending forward (eg, to tie shoes) (von Brevern, et al., 2007) (Furman, et al., 1999) (Dix, et al., 1952) (Whitney, et al., 2005).

Patients with BPPV most commonly report discrete, episodic periods of vertigo lasting 1 minute or less and often report modifications or limitations of their general movements to avoid provoking the vertiginous episodes (Ruckenstein, et al., 2007). Other investigators report that true “room spinning” vertigo is not always present as a reported symptom in posterior canal BPPV, with patients alternatively complaining of lightheadedness, dizziness, nausea, or the feeling of being “off balance” (Katsarkas, et al., 1999) (von Brevern, et al., 2007) (Herdman, et al., 1997) (Herdman, et al., 1997) (Macias, et al., 2000)(Cohen, et al., 2004) (Haynes, et al., 2002) (Blatt, et al., 2000) (Norre, et al., 1995). Approximately 50 percent of patients also report subjective imbalance between the classic episodes of BPPV (von Brevern, et al., 2007). In contrast, a history of vertigo *without* associated lightheadedness may increase the a priori likelihood of a diagnosis of posterior canal BPPV (Oghalai, et al., 2000). In up to one third of cases with atypical histories of positional vertigo, Dix-Hallpike testing will still reveal positional nystagmus, strongly suggesting the diagnosis of posterior canal BPPV (Norre, et al., 1995).

Other authors have loosened the historical criteria required for BPPV diagnosis with coinage of the term “subjective BPPV” without a positive Dix-Hallpike test (Haynes, et al., 2002) (Numez, et al., 2000). However, in clinical practice, there is a practical need to balance inclusiveness of diagnosis with accuracy of diagnosis.

Table 2.2 Diagnostic criteria for definitive, probable and atypical p-BPPV.

	History	Dix Hallpike maneuver	
		Vertigo	Nystagmus
Definitive p-BPPV	+	+	+
Probable p-BPPV	+	+	-
Possible (historical) p-BPPV	+	-	-

* NB. A negative Dix-Hallpike does not exclude a h-BPPV, see below.

Conclusie

Niveau 2	De diagnose posterieur kanaal BPPD (p-BPPD) is 'zeker' als sprake is van episodische symptomen van positionele draaiduizeligheid en een positieve Dix-Hallpike manoeuvre. Er is sprake van een 'waarschijnlijke' p-BPPD als de anamnese typisch is voor p-BPPD en de Dix Hallpike wel draaiduizeligheid uitlokt maar geen nystagmus. P-BPPD is 'mogelijk', als de anamnese typisch is voor p-BPPD, maar de Dix-Hallpike manoeuvre is negatief ten aanzien van draaiduizeligheid en nystagmus.

Physical examination

In addition to the historical criteria for the diagnosis of posterior canal BPPV, clinicians should confirm the diagnosis of posterior canal BPPV by performing the Dix-Hallpike maneuver (Table 2.1, Fig 2.1). The nystagmus produced by the Dix-Hallpike maneuvers in posterior canal BPPV typically displays two important diagnostic characteristics. First, there is a latency period between the completion of the maneuver, and the onset of subjective rotational vertigo and the objective nystagmus.

The latency period for the onset of the nystagmus with this maneuver is largely unspecified in the literature, but the panel felt that a typical latency period would range from 5 to 20 seconds, although it may be as long as 1 minute in rare cases (Baloh, et al., 1987). Second, the provoked subjective vertigo and the nystagmus increase, and then resolve within a time period of 60 seconds from the onset of nystagmus.

The fast component of the nystagmus provoked by the Dix-Hallpike maneuver demonstrates a characteristic mixed torsional and vertical movement (often described as upbeating-torsional), with the upper pole of the eye beating toward the dependent ear and the vertical component beating toward the forehead (Fig 2.1) (Furman, et al., 1999) (Honrubia, et al., 1999). Temporally, the rate of nystagmus typically begins gently, increases in intensity, and then declines in intensity as it resolves. This has been termed crescendo-decrescendo nystagmus. The nystagmus is again commonly observed after the patient returns to the upright head position and upon arising, but the direction of the nystagmus may be reversed.

Another classical feature of the nystagmus associated with posterior canal BPPV is that the nystagmus typically fatigues (a reduction in severity of nystagmus) when the maneuver is repeated (Dix, et al., 1952) (Honrubia, et al., 1999). However, repeated performance of the Dix-Hallpike maneuver to demonstrate fatigability is not recommended, because it unnecessarily subjects patients to repeated symptoms of vertigo that may be discomforting, and repeat performance may interfere with the immediate bedside treatment of BPPV (Furman, et al., 1999). Therefore, the panel did not include fatigability of the nystagmus as a diagnostic criterion.

Performing the Dix-Hallpike Diagnostic Maneuver

The Dix-Hallpike maneuver is performed by the clinician moving the patient through a set of specified head-positioning maneuvers to elicit the expected characteristic nystagmus of posterior canal BPPV (Fig 2.1) (Furman, et al., 1999) (Dix, et al., 1952). Before beginning the maneuver, the clinician should counsel the patient regarding the upcoming movements and warn that they may provoke a sudden onset of intense subjective vertigo, possibly with nausea, which will subside within 60 seconds. Because the patient is going to be placed in the supine position relatively quickly with the head position slightly below the body, the patient should be oriented so that, in the supine position, the head can "hang" with support off the posterior edge of the examination. The examiner should ensure that he can support the patient's head and guide the patient through the maneuver safely and securely, without the examiner losing support or balance himself.

1. The maneuver begins with the patient in the upright seated position with the examiner standing at the patient's side (Furman, et al., 1999). If present, the patient's eyeglasses should be removed. We initially describe the maneuver to test the right ear as the source of the posterior canal BPPV.
2. The examiner rotates the patient's head 45 degrees to the right and, with manual support, maintains the 45-degree head turn to the right during the next part of the maneuver.
3. Next, the examiner fairly quickly moves the patient (who is instructed to keep the eyes open) from the seated to the supine right-ear down position and then extends the patient's neck slightly (approximately 20 degrees below the horizontal plane) so that the patient's chin is pointed slightly upward, with the head hanging off the edge of the examining table and supported by the examiner. The examiner observes the patient's eyes for the latency, duration, and direction of the nystagmus (Norre, et al., 1988) (White, et al., 2005). Again, the provoked nystagmus in posterior canal BPPV is classically described as a more or less mixed torsional movement with the upper pole of both eyes beating toward the affected ear (in this example the right ear) in combination with a vertical (upbeat) component. The patient should also be queried as to the presence of subjective vertigo.
4. After resolution of the subjective vertigo and the nystagmus, if present, the patient may be slowly returned to the upright position. During the return to the upright position, a reversal of the nystagmus may be observed and should be allowed to resolve (a torsional nystagmus to the healthy ear, in combination with a vertical (downbeat) component).
5. The Dix-Hallpike maneuver (steps 1-4) should then be repeated for the left side, with the left ear arriving at the dependent position (Numez, et al., 2000). Again, the examiner should inquire about subjective vertigo and identify objective nystagmus, when present. The examination of the left side completes the test. The provoked nystagmus in left ear posterior canal BPPV is more or less mixed torsional movement with the upper pole of both eyes beating toward the affected ear (in this example the left ear) in combination with a vertical (upbeat) component. The Dix-Hallpike maneuver is considered the gold standard test for the diagnosis of posterior canal BPPV (Fife, et al., 2008). It is the most common diagnostic criterion required for entry into clinical trials and for inclusion of such trials in meta-analyses`(Hilton, et al., 2004) (Cohen, et al., 2005). The lack of an alternative external gold standard to the Dix Hallpike maneuver limits the availability of rigorous sensitivity and specificity data. Although it is considered the gold standard test for posterior canal BPPV diagnosis, its accuracy may differ between specialty and nonspecialty clinicians. Lopez-Escamez et al (Lopez-Escamez, et al., 2000) have reported a sensitivity of 82 percent and specificity of 71 percent for the Dix-Hallpike maneuvers in posterior canal BPPV, primarily among specialty clinicians. In the primary care setting, Hanley and O'Dowd (Hanley, et al., 2002) have reported a positive predictive value for a positive Dix-Hallpike test of 83 percent and a negative predictive value of 52 percent for the diagnosis of BPPV. Therefore, a negative Dix-Hallpike maneuver does not necessarily rule out a diagnosis of posterior canal BPPV. Because of the lower negative predictive values of the Dix-Hallpike maneuver, it has been suggested that this maneuver may need to be repeated at a separate visit to confirm the

diagnosis and avoid a false-negative result (Numez, et al., 2000) (Viire, et al., 2005) (Norre, et al., 1994).
Factors that may affect the diagnostic accuracy of the Dix-Hallpike maneuver include the speed of movements during the test, time of day, and the angle of the plane of the occiput during the maneuver (Numez, et al., 2000). The Dix-Hallpike test must be done bilaterally to determine which ear is involved or if both ears are involved (Numez, et al., 2000). In a small percent of cases, the Dix-Hallpike maneuver may be bilaterally positive (ie, the correspondingly appropriate nystagmus is elicited for each ear in the dependent position). For example, bilateral posterior canal BPPV is more likely to be encountered after head trauma (Katsarkas, et al., 1999).
Although the Dix-Hallpike maneuver is the test of choice to confirm the diagnosis of posterior canal BPPV, it should be avoided in certain circumstances. Although there are no documented reports of vertebrobasilar insufficiency provoked by performing the Dix-Hallpike maneuver, clinicians should be careful to consider the risk of stroke or vascular injury in patients with significant vascular disease (Whitney, et al., 2006). Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity (Whitney, et al., 2005) (Whitney, et al., 2006). Patients who are obese may be difficult for a single examiner to fully support throughout the maneuver, so additional assistance may be required. For patients with physical limitations, special tilting examination tables may allow the safe performance of the Dix-Hallpike maneuver.
To our knowledge, no comparative studies have been performed so far to investigate whether the diagnostic accuracy of the Hallpike maneuver with observation of the nystagmus by the naked eye improves by the use of Frenzel's glasses or infra-red Video-Oculography.

Conclusies

Niveau 2	De Dix-Hallpike manoeuvre is de gouden standaard is om posterieur kanaal BPPD te diagnosticeren.

Niveau 4	Het is niet aangetoond, maar het lijkt waarschijnlijk dat video-oculografie behulpzaam is bij het interpreteren van de nystagmus en biedt het voordeel dat documentatie van de nystagmus mogelijk is. De experts zijn van mening dat voor het beoordelen van de nystagmus een Frenzel bril niet strikt noodzakelijk is. *Niveau D: bronnen (niet-vergelijkend onderzoek Jackson et al 2007, en case studie Bertholon 2002))*

Overwegingen

- Voordeel: duidelijkheid omtrent de diagnose
- Nadelen: het mogelijk provoceren van draaiduizeligheid
- Kosten: minimaal
- Afweging van voordeel tegen nadeel: het voordeel weegt zwaarder.
- Waarde oordelen: Dix-Hallpike manoeuvre is de gouden standaard testmethode voor het stellen van de diagnose BPPD
- Rol van de voorkeur van de patiënt: minimaal.
- Exclusie: patiënten met fysieke beperkingen van de nek, zoals ernstige reumatoïde arthritis en cervicale radiculopathie.

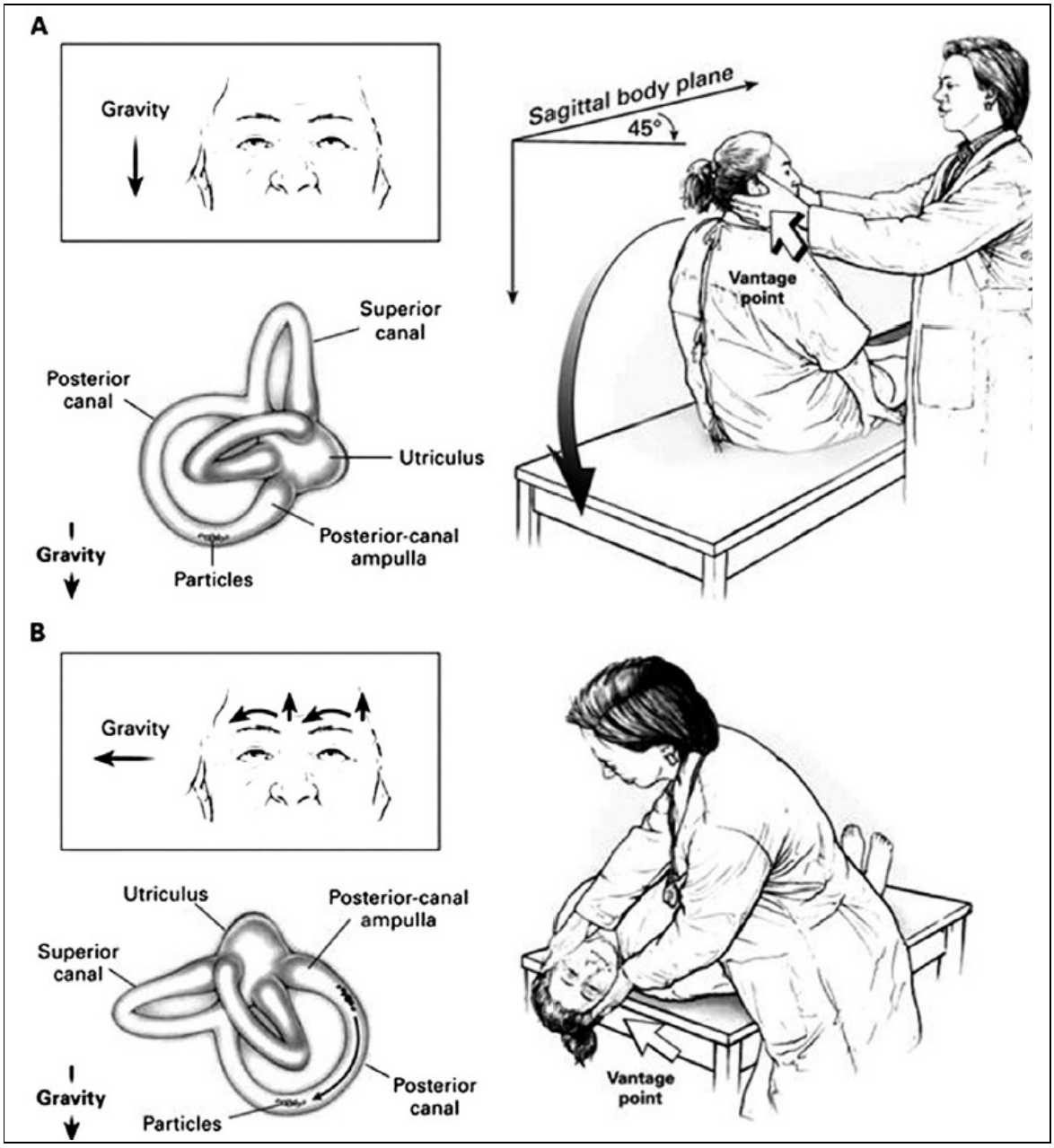

Figure 2.1

Diagrammatic representation of performance of the Dix-Hallpike maneuver for the diagnosis of posterior canal BPPV (adapted from reference 28). (**A**) The examiner stands at the patient's right side and rotates the patient's head 45 degrees to the right to align the right posterior semicircular canal with the sagittal plane of the body. (**B**) The examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The *arrows* in the inset depict the

direction of nystagmus in patients with typical benign paroxysmal positional vertigo. A presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.

Aanbeveling

De diagnose BPPD van het posterieur kanaal (zekere BPPD) wordt gesteld wanneer draaiduizeligheid met nystagmus wordt opgewekt door de Dix-Hallpike manoeuvre.

1B Diagnosis of Horizontal canal BPPD

Uitgangsvraag 1B:

Wat is de beste manier om BPPD van het horizontale kanaal (h-BPPD) te diagnosticeren?

Lateral canal BPPV (also called horizontal canal BPPV) is the second most common type of BPPV (Imai, et al., 2005) (Steenerson, et al., 2005) (Moon, et al., 2006). Because this type of BPPV has received considerably less attention in the literature, clinicians may be relatively unaware of its existence and the appropriate diagnostic maneuvers for lateral canal BPPV. Patients with a history compatible with BPPV (ie, repeated episodes of vertigo produced by changes in head position relative to gravity) who do not meet diagnostic criteria for posterior canal BPPV should be investigated for lateral canal BPPV. In many instances, the presenting symptoms of lateral canal BPPV are indistinguishable from posterior canal BPP (Streenerson, et al., 2005). Several studies have cited an incidence of approximately 10 to 15 percent in populations referred for evaluation and treatment of BPPV (White, et al., 2005) (Cakir, et al., 2006) (Hornibrook, et al., 2004) (Han, 2006) (Caruso, et al., 2005). Furthermore, lateral canal BPPV may occur following performance of the PRMs (eg, Epley maneuver) for an initial diagnosis of posterior canal BPPV. This transition from posterior canal BPPV to lateral canal BPPV is thought to occur as free-floating particulate material migrates from the posterior canal to the lateral canal (so-called canal switch). Because this type of transition is relatively common, clinicians should be aware of lateral canal BPPV and its diagnosis (White, 2005). The supine roll test is the preferred maneuver to diagnose lateral canal BPPV (Cakir, et al., 2006) (Moon, et al., 2006) (Nuti, et al., 1998). Clinicians should inform the patient that this test is a provocative maneuver and may cause the patient to become subjectively intensely dizzy for a short period of time. The supine roll test is performed by initially positioning the patient supine with the head in neutral position followed by quickly rotating the head 90 degrees to one side with the clinician observing the patient's eyes for nystagmus (Fig 2.2). After the nystagmus subsides (or if no nystagmus is elicited), the head is then returned to the straight faceup supine position. After any additional elicited nystagmus has subsided, the head is then quickly turned 90 degrees to the opposite side, and the eyes are once again observed for nystagmus. Two potential nystagmus findings may occur with this maneuver, reflecting two types of lateral canal BPPV (White, et al., 2005) (Nuti, et al., 1998) (Tirelli, et al., 2004).

- *Geotropic type:* In most cases of lateral canal BPPV, rotation to the affected side causes a very intense horizontal nystagmus beating toward the undermost (affected) ear, known as geotropic nystagmus (ie, nystagmus with a fast component toward the ground). When the patient is rolled to the other, healthy side, there is a less intense horizontal nystagmus, again beating toward the undermost ear (again geotropic; the direction of the nystagmus has now changed).
- *Apogeotropic type:* In less common cases, rotation to the healthy side results in an intense horizontal nystagmus beating toward the uppermost (affected) ear, known as anapogeotropic nystagmus (ie. nystagmus with a fast component away from the ground). Upon rolling to the opposite (affected) side, the nystagmus will change direction, beating toward the uppermost (healthy) ear, however less intense.

In both types of lateral canal BPPV, the fast component of the strongest nystagmus always beats to the affected ear (Han, et al., 2006) (Nuti, et al., 1998) (Baloh, et al., 1993). Between the two types of lateral canal BPPV, the geotropic variant predominates (Steenerson, et al., 2005) (Nuti, et al., 1998) (Casani, et al., 2002). Not uncommonly, because of CNS adaptation, the initially intense nystagmus may spontaneously change direction without rolling toward the opposite ear (Tirelli, et al., 2004). Also, in case of canalolithiasis, the position of the clod in the canal determines whether a geotropic (close to the utriculus) or apo-geotropic (close to the cupula) nystagmus will be induced as well,

which may lead to a “spontaneous” transition from geotropic into apo-geotropic or vice-versa (Califano L, et al., 2010).
The supine roll test has not received as much widespread use or diagnostic validation as the Dix-Hallpike maneuver. Review of the literature reveals that the sensitivity and specificity of the supine roll test in the diagnosis of lateral canal BPPV have not been determined. The lack of a more accurate, commonly accepted (gold standard) test for the diagnosis of lateral canal BPPV may be responsible, in part, for the absence of data for these statistical measures. A positive supine roll test, however, is the most commonly required and consistent diagnostic entry criterion for therapeutic trials of lateral canal BPPV (Steenerson, et al., 2005) (Han, et al., 2006). Reports of harm or patient injury from the performance of the supine roll test were not identified in the literature review, although many authors simply stated that patients who could not tolerate positional maneuvers were excluded from the population under study. Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, limited cervical range of motion, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget’s disease, ankylosing spondylitis, low back dysfunction, spinal cord injuries, and morbid obesity (Whitney, et al., 2005) (Whitney, et al., 2006). The benefit of performing the supine roll test is that it allows clinicians to confirm a diagnosis of lateral canal BPPV quickly and efficiently (White, et al., 2005) (Fife, et al., 2008). It also allows clinicians to more accurately and comprehensively diagnose positional vertigo that is not due to the posterior canal, whereas without supine roll testing, patients with lateral canal BPPV might be diagnostically missed if only traditional Dix-Hallpike testing was done. Further benefit might be derived from the supine roll test by decreasing the need to perform potentially unnecessary or unhelpful diagnostic testing.

Conclusie

Niveau 3	De diagnose horizontale kanaal BPPD wordt gesteld wanneer draaiduizeligheid met horizontale nystagmus wordt opgewekt door de supine roll test.

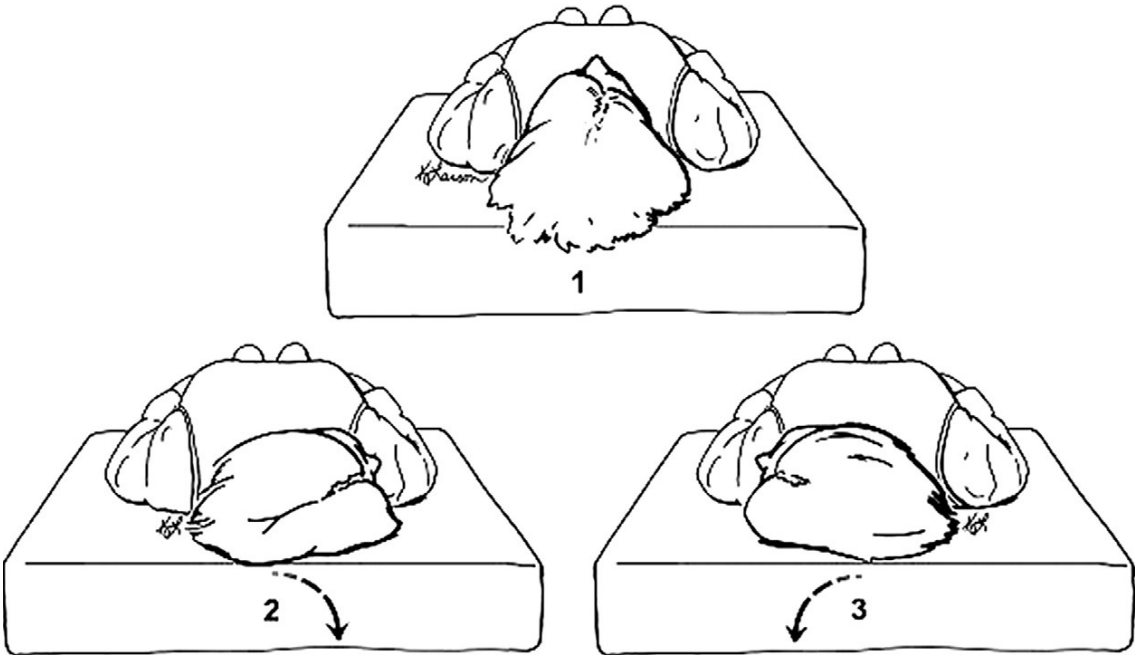

Figure 2.2
Diagrammatic views of the supine roll test. (**1**) The patient is in the starting neutral position. The patient's head is turned rapidly to the right side (**2**) to examine for characteristic nystagmus. Then the head is returned to the face-up position (**1**), allowing all nystagmus to subside, and then turned rapidly to the left side (**3**) to examine once again for nystagmus. (*From Fife et al., 2008).*

Overwegingen

- Voordeel: het onderkennen van een horizontale kanaal BPPD bij een negatieve Dix-Hallpike.
- Nadelen: het mogelijk provoceren van draaiduizeligheid
- Kosten: minimaal
- Afweging van voordeel tegen nadeel: het voordeel weegt zwaarder.
- Waarde oordelen: het belang van het onderzoeken van de patiënt op andere vormen van BPPD dan posterieur kanaal BPPD.
- Rol van de voorkeur van de patiënt: minimaal.
- Exclusie: patiënten met fysieke beperkingen

Aanbeveling

Als de patient een anamnese heeft die past bij BPPD maar de Dix-Hallpike is negatief, moet een BPPD van het horizontale kanaal worden overwogen en een supine roll test worden gedaan.

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Uitgangsvraag1c:
Wat is de beste manier om BPPD van het anterieure kanaal (a-BPPD) te herkennen?

Introduction
Benign paroxysmal positional vertigo (BPPV) of the anterior semicircular canal (ASC) is very rare. This is probably due to the orientation of the anterior canal in the head; gravity restricts the upward movement of the statoconien debris, preventing it from entering in the canal. If debris enters the canal, gravity facilitates self-clearance through the posterior arm of the ASC into the common crus and vestibule (Korres et al., 2002). An exception is cupulolithiasis, in which debris is fixed to the cupula and can not easily leave the canal simply by gravity or 'mass inertia'.
Some studies, that did not objectivate the nystagmus with EOG or VOG, show that anterior canal types of BPPV constitued 1.6 to 12% of the cases (Celebisoy et.al., 2008, Cakir et.al., 2006, Korres et.al., 2007, Korres et.al., 2002). One study, based on EOG findings, showed a downbeat nystagmus pointing to an anterior canal BPPV in 20% of cases. This suggests that objectivating nystagmus is important for adequately diagnosing anterior canal BPPV. The optimal method to do this clinically is using infrared videorecording (Ir-VOG) that allows repeated analysis of the nystagmus type and direction without fatigueing the response by repetitive positioning manoevres. Anterior canal BPPV must be differentiated from central-downbeat positional nystagmus.

Literature summary
In the literature search we found seven case studies that described the clinical signs of anterior canal BPPV (Bertholon et.al., 2002) (Imai et.al., 2006) (Korres et.al., 2008) (Lopez-Escamez, et.al., 2006) (Ogawa et.al., 2009) (Walther et.al., 2008) (Zapala et.al., 2008). In addition we found 1 non-systematic review that described the clinical signs of BPPV (Korres et.al., 2010).

Anterior canal BPPV
The Dix-Hallpike test stimulates the contralateral anterior canal that is located in the uppermost ear during the test. In anterior canal canalithiasis, BPPV is typically characterized by a
predominantly down-beating nystagmus with a small torsional component during Dix-Hallpike testing. The possibly small torsional component may be more pronounced when the patient looks in the direction of the undermost healthy ear and is clockwise in case of a canalolithiasis of the right anterior canal and anti-clockwise in case of a canalolithiases of the left anterior canal.
(Bertholon et.al., 2002, Korres et.al., 2006, 2008). Some authors report that the torsional component is often very small or absent. The direction of the torsional component of the nystagmus and the side on which vertigo and nystagmus are provoked are very important elements that may point at the affected ear. Diagnosing the affected ear is important for treatment of the affected ear (Bertholon et al., 2002), Korres et.al., 2006, 2008).
As an alternative for the Dix-Hallpike maneuver the 'straight head-hanging' provocation maneuvre is described. The head of a patient is in a middle position and the patient is placed with the head in retroflexion, stimulating both anterior canals simultaneously. Because the head is more extended during the straight head hanging position than during the Dix-Hallpike test sometimes a nystagmus may be triggered with the straight head hanging position, whereas the Dix-Hallpike test was negative. The provoked nystagmus is mostly downbeat without a clear torsional component, which makes it difficult to atribute the canalolithiases to right or left anterior canal (Korres et al, 2008; Lopez-Escamez et al., 2006).

Conclusies

Niveau 2/3	Bij patiënten met een typische anamnese voor BPPD, en een downbeat nystagmus bij de Dix Hallpike, moet de diagnose anterieur kanaal BPPD of centrale positioneringsnystagmus worden overwogen. *bronnen (?)*

Niveau 4	Het is niet aangetoond, maar het lijkt waarschijnlijk dat video-oculografie behulpzaam is bij het interpreteren van de verticale component van de nystagmus. *Niveau D: bronnen (niet-vergelijkend onderzoek Jackson et al 2007, en case studie Bertholon 2002))*

Overwegingen

- Voordeel: toegenomen diagnostische accuratesse en efficiëntie.
- Nadelen: Het tijdelijk provoceren van BPPD symptomen
- Kosten: minimaal
- Afweging van voordeel tegen nadeel: de aandoening is zeer zeldzaam, zodat de procedures voor diagnostiek van anterieur kanaal BPPD niet routinematig worden uitgevoerd.
- Rol van de voorkeur van de patiënt: matig.
- Exclusie: patiënten met fysieke beperkingen zoals ernstige reumatoïde artritis en cervicale radiculopathie.

Aanbeveling

De diagnose a-BPPD wordt gesteld wanneer draaiduizeligheid met downbeat nystagmus wordt opgewekt door de Dix-Hallpikemanoevre.

Tabel 2.2: criteria gebruikt voor de identificatie van het semicirculaire kanaal en de gepaste behandeling

Nystagmus/maneuver	Nystagmus duur (s)	Type	Aangedane semiciruclaire kanaal
Upbeat and anti-clockwise / right Dix-Hallpike	<60	Canalolithiasis	Rechter posterior kanaal
	>60	Cupulolithiasis (Rare)	
Upbeat and clockwise / left Dix-Hallpike	<60	Canalolithiasis	Linker posterior kanaal
	>60	Cupulolithiasis (Rare)	
Downbeat and clockwise / right Dix-Hallpike	<60	Canalolithiasis	Rechter anterior kanaal
	>60	Cupulolithiasis	
Downbeat en anti-horair na linker Dix-Hallpike	<60	Canalolithiasis	Linker anterior kanaal
	>60	Cupulolithiasis	
Horizontaal geotroop/ roll test; nystagmus het sterkst na roll naar rechts.	<60	Canalolithiasis	Rechter horizontaal kanaal
Horizontaal apogeotroop/ roll test; nystagmus het sterkst na roll naar rechts.	>60	Cupulolithiasis	
Horizontaal geotrooop/ roll test; nystagmus het sterkst na roll naar links.	<60	Canalolithiasis	Linker horizontaal kanaal
Horizontaal apogeotroop/ roll test; nystagmus het sterkst na roll naar links.	>60	Cupulolithiasis	

(Overgenomen uit: Anterior canal beniging paroxysmal positional vertigo, Jackson et al, 2007, die hebben het aangepast en overgenomen uit Herdman SJ Advances in the treatment of vastibular disorders. Phys Ther 1997; 77: 602-18)

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Verklarende Woordenlijst

Otoconia (Thalmann, et al., 2001) zijn kleine kristallen van calcium carbonaat in de oorblaasjes (deel van het vliezige vestibulaire apparaat, gelegen tegen de binnenwand van de voorhof) en utriculus (het gedeelte van het membraneuze labyrinth van het oor waarin het semicirculaire kanaal opent) van het oor die onder de invloed van een versnelling in een rechte lijn de haarcellen stimuleren door hun beweging ten opzichte van het gelatineuze ondersteunende substraat waarin de cilia van de haarcellen ingebed zijn.)

Otoconia: small crystals of calcium carbonate in the saccule and utricle of the ear that under the influence of acceleration in a straight line cause stimulation of the hair cells by their movement relative to the gelatinous supporting substrate containing the embedded cilia of the hair cells—called also statoconia

Hoofdstuk 3 Differentiaal diagnose BPPD

Uitgangsvraag 2:

Van welke andere vormen van positioneringsduizeligheid moet BPPD worden onderscheiden?

Despite being the most common cause of peripheral vertigo, (Froehling, et al., 2000) BPPV is still often underdiagnosed or misdiagnosed (von Brevern, et al., 2004). Causes of vertigo that may be confused with BPPV can be divided into otological, neurological, and other entities.. In subspecialty settings, BPPV caused 10.1% of vertigo, while neurological causes were rare (Bruintjes, et al., 2007).

The most common diagnoses that require distinction from BPPV are listed in Table 3.1. These conditions require distinction from BPPV because their natural history, treatment, and potential for serious medical sequelae differ significantly.

Otologic disorders

Other otological disorders causing vertigo may be differentiated from BPPV by their clinical characteristics including their temporal pattern and the presence or absence of hearing loss. Whereas BPPV is characterized by acute, discrete episodes of brief positional vertigo without associated hearing loss, other otological causes of vertigo manifest different temporal patterns and may additionally demonstrate associated hearing loss (Kentala, et al., 2003). In distinction to BPPV, Ménière's disease is characterized by discrete episodic attacks, with each attack exhibiting a characteristic triad of sustained vertigo, fluctuating hearing loss, and tinnitus (Baloh, et al., 1987), (Wladislavosky-Waserman, et al., 1984). Recurrent vestibulopathy is characterized by similar episodic attacks of vertigo, but lacks any auditory symptoms (van Leeuwen et al., 2010).
As opposed to BPPV, the duration of vertigo in an episode of Ménière's disease or recurrent vestibulopathy typically lasts longer (usually on the order of hours) and is typically more disabling owing to both severity and duration. In addition, an associated contemporaneous decline in sensorineural hearing is required for the diagnosis of a Ménière's attack, whereas acute hearing loss should not occur with an episode of BPPV (Thorp, et al., 2003). Protracted nausea and vomiting are also more common during an attack of Ménière's disease or recurrent vestibulopathy.
Acute peripheral vestibular dysfunction syndromes, such as vestibular neuritis or labyrinthitis, present with sudden, unanticipated, severe vertigo with a subjective sensation of rotational (room spinning) motion. If the auditory portion of the inner ear is affected, hearing loss and tinnitus may also result (Baloh, et al., 2003). These syndromes are commonly preceded by a viral prodrome. The time course of the vertigo is often the best differentiator between BPPV and vestibular neuritis or labyrinthitis. In vestibular neuritis or labyrinthitis, the vertigo is of gradual onset, developing over several hours, followed by a sustained level of vertigo lasting days to weeks (Kentala, et al., 2003) (Kentala, et al., 1996) (Kentala, et al., 1999). The vertigo is present at rest (not requiring positional change for its onset), but it may be subjectively exacerbated by positional changes. These acute peripheral vestibular syndromes may also be accompanied by severe levels of nausea, vomiting, sweating, and pallor, which are also typically sustained along with the vertigo.

Tabel 3.1

Differentiaaldiagnose BPPD

Otologische stoornissen	Neurologische stoornissen	Andere oorzaken
Ziekte van Menière	Vestibulaire migraine	Angst of paniekstoornissen
Recurrent vestibulopathy	Vertebrobasilaire TIA's	Orthostatische hypotensie
Neuritis vestbularis		
Labyrinthitis	Demyeliniserende ziektes (MS)	Bijwerkingen van medicijnen
Superior canal dehiscence syndrome	Centraal zenuwstelsel lesies	
Posttraumatische vertigo		

Superior canal dehiscence syndrome (SCD) is clinically characterized by attacks of vertigo and oscillopsia (the sensation that viewed objects are moving or wavering back and forth) often brought on by loud sounds, Valsalva maneuvers, or pressure changes of the external auditory canals (Minor, et al., 2001). Similar to perilymphatic fistula, it differs from BPPV in that vertigo is induced by pressure changes and not position changes. SCD may also present with an associated conductive hearing loss and is diagnosed through CT of the temporal bones (Rosowski, et al., 2004).
Posttraumatic vertigo can present with a variety of clinical manifestations including vertigo, disequilibrium, tinnitus, and headache (Marzo, et al., 2004). Although BPPV is most often idiopathic, in specific cases, traumatic brain injury is associated with BPPV (Davies, et al., 1995). BPPV has been described as occurring in conjunction with or as a sequelae to other vestibular disorders as well, such as Ménière's disease and vestibular neuritis (Karlberg, et al., 2000). Therefore, clinicians must consider the possibility of more than one vestibular disorder being present in any patient who does not clearly have the specific symptoms of a single vestibular entity.

Neurological disorders

One of the key issues facing clinicians attempting to diagnose the etiology for vertigo is the differentiation between peripheral causes of vertigo (those causes arising from the ear or vestibular apparatus) and CNS causes of vertigo. Although at times this distinction may be difficult, several clinical features may suggest a central cause of vertigo rather than BPPV (Labuguen, et al., 2006) (Baloh, et al., 1998). Nystagmus findings that more strongly suggest a neurological cause for vertigo, rather than a peripheral cause such as BPPV, include down-beating nystagmus on the Dix-Hallpike maneuver, direction-changing nystagmus occurring without changes in head position (ie, periodic alternating nystagmus), or baseline nystagmus manifesting without provocative maneuvers. Among the central causes of vertigo that should be distinguished from BPPV are migraine-associated vertigo, vertebrobasilar TIA, and intracranial tumors.
Vestibular migraine has been described as a common cause of vertigo in the adult population (Reploeg, et al., 2002) and may account for as many as 14 percent of cases of vertigo (Kentala, et al., 2003). Diagnostic criteria include 1) episodic vestibular symptoms; 2) migraine according to International Headache Society criteria; 3) at least two of the following migraine symptoms during at least two vertiginous episodes: migrainous headache, photophobia, phonophobia, or visual or other aura; and 4) other causes ruled out by appropriate investigations (Headache Classification Subcommittee of the International Headache Society, 2004). Migraine-associated vertigo is heterogeneous in that both central disorders and peripheral disorders have been described, although more often it is believed to be central in nature (Neuhauser, et al., 2001), (von Brevern, et al., 2005). It is distinguishable from BPPV by virtue of the necessary migraine/headache components, which are not associated with classic BPPV.

Several reports have suggested that isolated attacks of vertigo can be the initial and only symptom of vertebrobasilar insufficiency (Fife, et al., 1994), (Grad, et al., 1989) (Gomez, et al., 1996). Isolated transient vertigo may precede a stroke in the vertebrobasilar artery by weeks or months. The attacks of vertigo in vertebrobasilar insufficiency usually last less then 30 minutes and have no associated hearing loss. The type of nystagmus (typically gaze-evoked in central lesions), the severity of postural instability, and the presence of additional neurological signs are the main distinguishing features between vertebrobasilar insufficiency and BPPV (Gomez, et al., 1996) (Hotson, et al., 1998). In addition, the nystagmus arising in vertebrobasilar insufficiency does not fatigue and is not easily suppressed by gaze fixation, helping to separate this diagnosis from BPPV.
Intracranial tumors and other brain stem lesions may rarely present with a history and symptomatology similar to those of BPPV (Dunniway, et al., 1998). In these cases, associated symptoms such as tinnitus, aural fullness, new-onset hearing loss, and/or other neurological symptoms should help differentiate these diagnoses from BPPV. Atypical nystagmus during Dix-Hallpike testing (eg, sustained down-beating nystagmus) argues against BPPV and suggests a more serious cause. Finally, failure to respond to conservative management such as the PRM or vestibular rehabilitation should raise concern that the underlying diagnosis may not be BPPV (Dunniway, et al., 1998).

Other disorders

Several other non-otological and non-neurological disorders may present similarly to BPPV. Patients with panic disorder, anxiety disorder, or agoraphobia may complain of symptoms of lightheadedness and dizziness. Although these symptoms are usually attributed to hyperventilation, other studies have shown high prevalences of vestibular dysfunction in these patients (Jacob, et al., 1996) (Furman, et al., 2006). These conditions may also mimic BPPV. Several medications, such as Mysoline, carbamazepine, phenytoin, antihypertensive medications, and cardiovascular medications, may produce side effects of dizziness and/or vertigo and should be considered in the differential diagnosis.

Postural hypotension also may produce episodic dizziness or vertigo. The dizziness or vertigo in postural hypotension, however, is provoked by moving from the supine to the upright position in distinction to the provocative positional changes of BPPV.
Although the differential diagnosis of BPPV is vast, most of these other disorders can be further distinguished from BPPV on the basis of responses to the Dix-Hallpike maneuver and the supine roll test. Clinicians should still remain alert for concurrent diagnoses accompanying BPPV, especially in patients with a mixed clinical presentation.

Conclusie

Niveau 3	De differentiaaldiagnose van BPPD omvat alle aandoeningen die zich presenteren met houdingsafhankelijke duizeligheid. Hierbij moet vooral gedacht worden aan orthostatische hypotensie, centrale pathologie, angststoornissen (phobic postural vertigo) en vestibulaire migraine. Daarnaast dienen perifeer vestibulaire stoornissen, zoals bij voorbeeld M. Meniere, recurrent vestibulopathy, neuritis vestibularis en labyrinthitis, te worden overwogen.

Aanbeveling

BPPD moet met name gedifferentieerd worden van andere aandoeningen die zich presenteren met houdingsafhankelijke duizeligheid, zoals bijvoorbeeld orthostatische hypotensie en vestibulaire uitval.

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Hoofdstuk 4. Aanvullend onderzoek

Uitgangsvraag 3a:

Wat zijn de indicaties voor beeldvormend, audiologisch en vestibulair onderzoek bij verdenking op BPPD?

The diagnosis of BPPV is based on the clinical history and physical examination. Routine radiographic imaging or vestibular testing is unnecessary in patients who already meet clinical criteria for the diagnosis of BPPV (Table 2.1). Further radiographic or vestibular testing may have a role in the diagnosis if the clinical presentation is felt to be atypical, if Dix-Hallpike testing elicits equivocal or unusual nystagmus findings, or if additional symptoms aside from those attributable to BPPV are present, suggesting an accompanying modifying CNS or otological disorder.

Radiographic Imaging

Radiographic imaging, most commonly CNS imaging using magnetic resonance or CT techniques, is commonly obtained in the evaluation of a primary symptom complaint of vertigo. However, imaging is not useful in the routine diagnosis of BPPV because there are no radiological findings characteristic of or diagnostic for BPPV (Turski, et al., 1996) (Turski, et al., 1996). The lack of characteristic findings is likely due to fact that the pathology presumed to occur in BPPV within the semicircular canals occurs at a microscopic level that is beyond the resolution of current neuroimaging techniques (Parnes, et al., 1992). On a broader scale, previous retrospective reviews of elderly patients with dizziness failed to detect any significant differences in cranial MRI findings when comparing dizzy versus non-dizzy patients (Colledge, et al., 1996) (Day, et al., 1990).

Radiographic imaging of the CNS should be reserved for patients who present with a clinical history compatible with BPPV but who also demonstrate additional neurological symptoms atypical for BPPV. Radiographic imaging may also be considered for patients with suspected BPPV but inconclusive positional testing, or in patients with other neurological signs on physical examination that are not typically associated with BPPV. Such symptoms include abnormal cranial nerve findings, visual disturbances, and severe headache, among others. It should be noted that intracranial lesions causing vertigo are rare. (Hanley, et al., 2001) Potential lesions causing vertigo identifiable on CNS imaging include cerebrovascular disease, demyelinating disease, or an intracranial mass; they are most often located in the brain stem cerebellum, thalamus, or cortex (Hanley, et al., 2001). In small case series, positional vertigo and nystagmus have been associated with neurovascular compression of cranial nerve VIII, vestibular schwannoma, Arnold Chiari malformation, and a variety of cerebellar disorders (Brandt, et al., 1994) (Jacobsen, et al., 1995) (Kumar, et al., 2002).

In distinction to standard BPPV, such conditions are quite rare and typically present with additional neurological symptoms in conjunction with the vertigo. Routine neuroimaging has not been recommended to discern these conditions from the more common causes of vertigo (Gizzi, et al., 1996). The costs of routine imaging in cases of BPPV are not justified given that diagnostic neuroimaging does not improve the diagnostic accuracy in the vast majority of BPPV cases. Therefore, neuroimaging should not be routinely used to confirm the diagnosis of BPPV.

Vestibular Function Testing

When patients meet clinical criteria for the diagnosis of BPPV (Table 2.1), no additional diagnostic benefit is obtained from vestibular function testing. Vestibular function testing is indicated when the diagnosis of a vertiginous or dizziness syndrome is unclear or possibly when the patient remains symptomatic following treatment. It may also be beneficial when multiple concurrent peripheral vestibular disorders are suspected (Baloh, et al., 1987) (Kentala, et al., 1996)(Lopez-Escamez, et al., 2003).

Vestibular function testing involves a battery of specialized tests that primarily record nystagmus in response to labyrinthine stimulation and/or voluntary eye movements. Most vestibular function testing relies on the neurological relationship between the regulation of eye movement and the balance organs: the vestibular-ocular reflex. These tests are useful in the evaluation of vestibular disorders that may not be evident from the history and clinical examination, and may provide information for quantification, prognostication, and treatment planning (Gordon, et al., 1996). The components of the vestibular function test battery identify abnormalities in ocular motility as well as deficits in labyrinthine response to position change, caloric stimulation, rotational movement, and static positions (sitting and supine). Caloric testing is an established, widely accepted technique that is particularly useful in determining unilateral vestibular hypofunction. Rotational chair testing is considered the most sensitive and reliable technique for quantifying the magnitude of bilateral peripheral vestibular hypofunction (Fife, et al., 200) and to assess central compensation after peripheral vestibular loss. Some or all of these test elements may be included in a vestibular test battery.

In cases of BPPV in which the nystagmus findings are suggestive but not clear, it may be beneficial to use video-oculographic recordings of nystagmus associated with posterior canal BPPV. Especially video-recorded eye movements can be analysed in detail using image-processing techniques. for further study or second opinion without the need to repeat the Dix-Hallpike manoeuvre. A second diagnostic procedure often will result in more difficult to asses eye movements because of the typical fatigue of a BPPV. In a small percentage of cases, patients with a history of positional vertigo but unclear nystagmus findings may undergo vestibular function testing. Among complex patients referred for subspecialty evaluation of BPPV, such atypical or unclear nystagmus findings may approach 13 percent in patients with diagnoses suspicious for BPPV (Bath, et al., 2000).

BPPV is relatively frequently associated with additional vestibular pathology. Symptoms associated with chronic vestibular function may persist following appropriate treatment for BPPV, even if the treatment is effective in resolving the specific complaint of positional vertigo. For example, in highly selected subsets of patients referred for subspecialty evaluation of BPPV, additional otopathology and/or vestibulopathy has been identified in 31 to 53 percent of BPPV patients (Baloh, et al., 1987) (Roberts, et al., 2005) (Korres, et al., 2004). This percentage, however, is higher than what might be expected in the nonspecialty population. Vestibular disorders that have been associated with BPPV include Ménière's disease, viral vestibular neuritis, or labyrinthitis (Hughes, et al., 1997)(Karlberg, et al., 2000). Vestibular function testing may be obtained when these additional diagnoses are suspected on the basis of signs or symptoms in addition to those of BPPV.

In patients with vestibular pathology in addition to BPPV, PRMs appear to be equally effective in resolving the positional nystagmus associated with BPPV, but complete symptom resolution is significantly less likely in those patients with additional vestibular pathology. In one study, 86 percent of patients with BPPV but without associated vestibular pathology reported complete resolution of symptoms after PRMs versus only 37 percent reporting complete resolution when additional vestibular pathology was present (Pollak, et al., 2004).

Thus, patients with suspected associated vestibular pathology in addition to BPPV may be a subset who would benefit from the additional information obtained from vestibular function testing. Similarly, up to 25 percent of patients with separate recurrences of BPPV are more likely to have

associated vestibular pathology (Del Rio, et al., 2004); therefore, patients with recurrent BPPV may be candidates for vestibular function testing. In summary, patients with a clinical diagnosis of BPPV according to guideline criteria should not routinely undergo vestibular function testing, because the information provided from such testing adds little to the diagnostic accuracy in these cases, vestibular testing adds significant cost to the diagnosis and management of BPPV, and the information obtained does not alter the subsequent management of BPPV in the vast majority of the cases. Therefore, vestibular function testing should not be routinely obtained when the diagnosis of BPPV has already been confirmed by clinical diagnostic criteria. Vestibular function testing, however,may be warranted in patients with 1) atypical nystagmus, 2) suspected additional vestibular pathology, 3) a failed (or repeatedly failed) response to CRP, or 4) frequent recurrences of BPPV (Rupa, et al., 2004) (Gordon, et al., 2005).

Niveau 3	Beeldvormend en vestibulair onderzoek heeft geen toegevoegde waarde bij het stellen van de diagnose BPPD.

Overwegingen

- Voordeel: snelle behandeling mogelijk maken door het voorkomen van overbodige testen en voorkomen van mogelijke vals-positieve diagnoses; voorkomen van stralingsbelasting (MRI) en bijwerkingen door testen.
- Nadeel: potentieel missen van comorbide aandoeningen, ongemak door misselijkheid en braken ten gevolge van vestibulaire testen.
- Kosten: kostenbesparing door het voorkomen van overbodige testen.
- Afweging van voordeel tegen nadeel: het voordeel weegt zwaarder.
- Waarde oordeel: het is belangrijk om overbodige testen en vertraging in het stellen van de diagnose te voorkomen.

Aanbeveling 3a:

Beeldvormende technieken zijn niet geïndiceerd bij de diagnose BPPD. Beeldvormende technieken dienen wel te worden toegepast bij patiënten bij wie twijfel bestaat omtrent de diagnose BPP, bij voorbeeld als additionele neurologische uitvalssymptomen aanwezig zijn, of bij therapieresistente BPPD.

Vestibulaire functietesten hebben geen toegevoegde waarde bij patiënten met BPPD. Vestibulaire functietesten zijn alleen geïndiceerd bij patiënten met: 1) atypische nystagmus, 2) verdenking op additionele vestibulaire pathologie 3) een falende (of herhaaldelijk falende) reactie op canalith repositiemanoeuvres (CRM), of 4) frequent recidiverende BPPD.

Uitgangsvraag 3b:

Kunnen audiometrische testen de diagnose BPPD ondersteunen?

Audiometry is the most commonly obtained objective test of hearing. Recent Medicare data indicate that approximately 9 percent of audiograms obtained annually are ordered in association with diagnostic categories related to vertigo (International Classification of Diseases, Version 9 codes: 386 and/or 780.4) (American Medical association's relative value scale upgrade, 2008). Specialty clinicians with access to audiometry frequently obtain audiometry as part of the evaluation of vertigo in contradistinction to nonspecialty clinicians. However, limited diagnostic cohort studies and cost-effectiveness studies supporting this practice are available.

Audiometry is not required to diagnose BPPV; however, audiometry may offer some diagnostic benefit for patients in whom the clinical diagnosis of BPPV is unclear. Both hearing loss and BPPV are more prevalent in older patients. Therefore, BPPV and some degree of hearing loss (likely long-standing, as in presbyacousis) are likely to coexist in patients with BPPV (Havlik, et al., 1986). From a pathophysiological standpoint, a preexisting, stable hearing loss should be unrelated to and not influence the diagnosis of BPPV. In such cases, routine audiometry is unlikely to reinforce or influence the diagnosis of BPPV.

In the majority of cohort studies of BPPV, audiometric studies, when obtained, have been largely normal. In some of these studies, however, the inclusion criteria for a diagnosis of BPPV included no history of antecedent hearing loss (Kentala, et al., 2000). In two algorithmic studies, audiometry was found to be cost-effective and diagnostically effective in the broad evaluation of patients with vertigo (Kentala, et al., 2000) (Kentala, et al., 2003). In a study of 192 patients referred to an academic center for the evaluation of vertigo, Stewart et al (Stewart, et al., 1999) found that the audiogram was the most cost-effective test among various studies including electronystagmography, posturography, MRI, and blood tests. Notably, however, the cost-effectiveness (diagnostic benefit) of the history and physical examination (ie, Dix- Hallpike maneuver or supine role test) was not directly studied. This diagnostic focus notably differs from the current guideline, which emphasizes the value of the clinical history and physical examination.

In a study of 564 cases, Kentala et al (Kentala, et al., 1999) found in a diagnostic algorithm analysis that the presence of a normal audiogram was corroborating for a diagnosis of BPPV, distinguishing BPPV from other associated conditions such as Ménière's disease, vestibular schwannoma, and so on. However, the panel felt that distinction from such associated conditions could be made accurately and more cost-effectively on the basis of the history, rather than relying on audiometry. Upon review of the literature, no meaningful observational or diagnostic cohort studies either supporting or arguing against the use of audiometry in the diagnosis of the BPPV population was identified.

Traditional BPPV should not manifest with symptoms of a new-onset hearing loss. A newly reported hearing loss arising in conjunction with vertigo suggests a diagnosis other than BPPV and such patients merit audiometry. Clinicians should distinguish patients with vertigo and newonset hearing loss from those patients with preexisting ontological disease who subsequently develop BPPV. As noted, studies have reported rates of associated otological or vestibular pathology in 30 to 50 percent of cases in referred populations with BPPV (Baloh, et al., 1987) (Roberts, et al., 2005) (Korres, et al., 2004). In cases with preexisting otological disease and a diagnostic concern for BPPV, audiometry may help establish the independent stability of the otological disease, thereby helping to confirm a diagnosis of BPPV.

Audiometry is a noninvasive test with widespread availability and no reported harms from testing. The potential benefits of obtaining audiometry in the evaluation of BPPV include the ability to establish baseline stability or, alternatively, to help rule out other otological conditions such as Ménière's disease or labyrinthitis (Kentala, et al., 1999). The primary disadvantage of routinely obtaining audiometry in patients undergoing evaluation for BPPV is clearly the cost to the health care system. In the vast majority of cases of BPPV with stable hearing by history, the audiogram is most likely to be normal or demonstrate an age-appropriate sensorineural hearing loss and, therefore, likely will not influence the diagnosis of BPPV. Overall, insufficient evidence exists to either confirm or disaffirm the value of routine audiometry in the initial assessment of BPPV.

Conclusie 3b

Niveau 4	Audiometrie heeft geen toegevoegde waarde om BPPD te diagnosticeren.

Overwegingen

- Voordeel: geen vertraging in herkenning en behandeling van BPPD
- Nadeel: mogelijk missen van gehoorverlies (passend bij bijv. M. Meniere)
- Kosten: mogelijke realisatie van kostenbesparingen als er minder audiogrammen worden aangevraagd.
- Afweging van voordeel tegen nadeel: het voordeel weegt zwaarder.
- Waarde oordeel: Het is gemakkelijk om een kleine groep patiënten te identificeren waarbij audiometrie nuttig zou zijn op basis van de anamnese.
- Rol van de voorkeur van de patiënt: minimaal.

Aanbeveling 3B Audiometrisch testen

Er is bij BPPD geen indicatie voor het uitvoeren van audiometrisch onderzoek.

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Hoofdstuk 5 De behandeling van BPPD

Uitgangsvraag 4a:

Zijn repositiemanoeuvres geschikt als therapie om patiënten met BPPD te behandelen?

Although it has been historically commonplace to reassure patients diagnosed with BPPV that their condition is benign and is likely to spontaneously remit in the subsequent months, recent relatively high-quality evidence supports active, expeditious treatment with a particle repositioning maneuver (PRM). Treatment with PRMs consistently eliminates the vertigo due to BPPV, improves quality of life, and reduces the risks of falling.

Posterior Canal BPPV Treatments

Two types of PRMs have been found effective for posterior canal BPPV: 1) the canalith repositioning procedure (CRP, also referred to as the Epley maneuver) and 2) the liberatory maneuver (also called the Semont maneuver). Other PRMs have been proposed for the treatment of posterior canal BPPV, but high-quality, reproducible data that demonstrate their clinical efficacies are lacking.

Treatment with canalith repositioning procedure. CRP was first described by Epley in 1992 (Epley, et al., 1992). Through a series of head position changes, the CRP moves the canaliths from the posterior semicircular canal to the vestibule, thereby relieving the stimulus from the semicircular canal that had been producing the vertigo in BPPV. CRP is most commonly performed in the outpatient setting by a clinician after confirmation of the diagnosis of posterior canal BPPV (Fife, et al., 2008). Patients should be informed that nausea, occasional vomiting, and/or a sense of falling may arise during the CRP (Uneri, et al., 2005). Patients who previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuver may be considered for antiemetic prophylaxis during the CRP. Figure 5.1 depicts the CRP for posterior canal BPPV. Several RCTs have been published evaluating the efficacy of the CRP in the treatment of posterior canal BPPV. A number of these are high-quality RCTs, several of which have been included in a relatively recent Cochrane collaborative review of the Epley maneuver for BPPV (Hilton, et al., 2004) (Froehling, et al., 2000) (Lynn, et al., 1995) (Yimtae, et al., 2003). The Cochrane review identified a statistically significant effect in favor of the CRP compared with controls. An odds ratio of 4.2 (95% confidence interval, 2.0-9.1) was found in favor of treatment for subjective symptom resolution in posterior canal BPPV; an odds ratio of 5.1 (95% confidence interval, 2.3-11.4) was found in favor of treatment for conversion of a positive to negative Dix-Hallpike test. Another systematic review of randomized controlled trials provides strong evidence that the CRP resolves PC BPPV, and quasi-RCTs suggested that the CRP or the LM performed by a clinician or with proper instruction at home by the patient resolves PC BPPV (Helminski, et al., 2010). This systematic review included no data on the effects of the maneuvers on outcomes relevant to patients (Helminski, et al., 2010). Waleem et. al. conducted an RCT that compared an Epley maneuver with placebo treatment and found that 82% of patients in the Epley group was cured after two weeks, significantly more (P=0.002) than the 32% placebo group (Waleem, et al., 2008). Therefore, Epley's maneuver is a much better form of management for benign paroxysmal positional vertigo than expectant treatment.

Subsequently, additional RCTs have been published regarding the CRP, reflecting similar results. Table 5.1 summarizes recent RCTs evaluating CRP for posterior canal BPPV. Of note, consistent with the expected spontaneous resolution of posterior canal BPPV over time, treatment effects between CRP and control patients tended to diminish over time. In the short term, typically at 1 week, the CRP is very effective at providing symptom resolution for posterior canal BPPV with small numbers needed to treat (NNT).

All but one of the RCTs for CRP has taken place in the specialized clinic setting, most commonly with a referred population, which may limit the generalizability of these results. In the only RCT conducted in the primary care setting, investigators were unable to demonstrate a significant benefit for the CRP based on symptomatic outcome (Munoz, et al., 2007). At 1 week follow-up, 31.6 percent (12/38) of CRP patients demonstrated symptom resolution versus 24.4 percent (10/ 41) of sham patients (*P* _ 0.48). Objectively, however, 34.2 percent of CRP-treated patients converted to a negative Dix-Hallpike at 1 week, versus 14.6 percent in the sham group (*P* _ 0.04). Although statistically significant, this objective conversion rate is still lower than those reported among RCTs in the specialty setting (typically ranging from 66%-89%) (Hilton, et al., 2004). Because both the symptomatic response rates and conversion rates to a negative Dix-Hallpike maneuver are lower than those reported in specialty setting RCTs, further investigation into the effectiveness of the CRP in the primary care setting is warranted. Reasons for discrepancy between primary care and specialty settings may include differences in performance of the CRP (ie, a single maneuver vs repeated maneuvers at the same visit), intrinsic patient variability with comorbid balance disorders, differences in symptom reporting, or combinations thereof. In a Randomized, controlled, prospective trial. A Dizzy Fix score was used for home treatment of BPPV. The Dizzy Fix assisted users in performing a PRM and Dizzy Fix users performed significantly better on their PRM (particle replacement Manoevre) performance compared with controls (p = .0001). Results are promising and now we are waiting for results of a clinical trial using the Dizzy Fix in a group of patients. This seems a significant improvement from written instructions or in-office training. (Bromwich, et al., 2008) The positive treatment results of the CRP have also been demonstrated in lesser quality nonrandomized trials and case series (Sherman, et al., 2001) (Li, et al., 1995) (Lempert, et al., 1997) (Wolf, et al., 1999) (Asawavichianginda, et al., 2000) (Angeli, et al., 2003) (Chang, et al., 2004). In addition to the Cochrane review, four meta-analyses have been reported (White, et al., 2005) (Lopez-Escamez, et al., 1999) (Woodworth, et al., 2004) (Teixeira, et al., 2006). Each analysis concluded that the CRP is significantly more effective than placebo in posterior canal BPPV. Among these trials, however, significant heterogeneity has also been demonstrated (Teixeira, et al., 2006).

Many trials also report a secondary outcome of conversion from a positive to negative Dix-Hallpike maneuver after CRP. The odds ratios for this more objective measure of resolution for posterior canal BPPV range from 3.2 to 22 across studies, similar to reported rates of symptom resolution (Hilton, et al., 2004). In most nonrandomized case series assessing treatment response, symptom resolution is the only commonly reported outcome measure for the CRP.

Considerable variability exists in terms of the number of times the CRP is applied for the initial treatment of BPPV, even across RCTs (Froehling, et al., 2000) (Lynn, et al., 1995) (Yimtae, et al., 2003). Some investigators perform only one CRP cycle at the initial treatment, whereas others repeat a fixed number of cycles or perform the CRP repeatedly until the vertiginous symptoms extinguish or the Dix- Hallpike converts to negative (Lynn, et al., 195). Even further variability exists among published case series for CRP (Ruckenstein, et al., 2001) (Sekine, et al., 2006) (Prokopakis, et al., 2005). On the basis of a review of the literature, it was not possible to determine the optimal number of cycles for the CRP or a protocol for repeated procedures. The repeated application of the CRP is

likely to be determined by the severity of the symptoms, if they persist; clinician availability; and the clinician's historical success with the CRP.

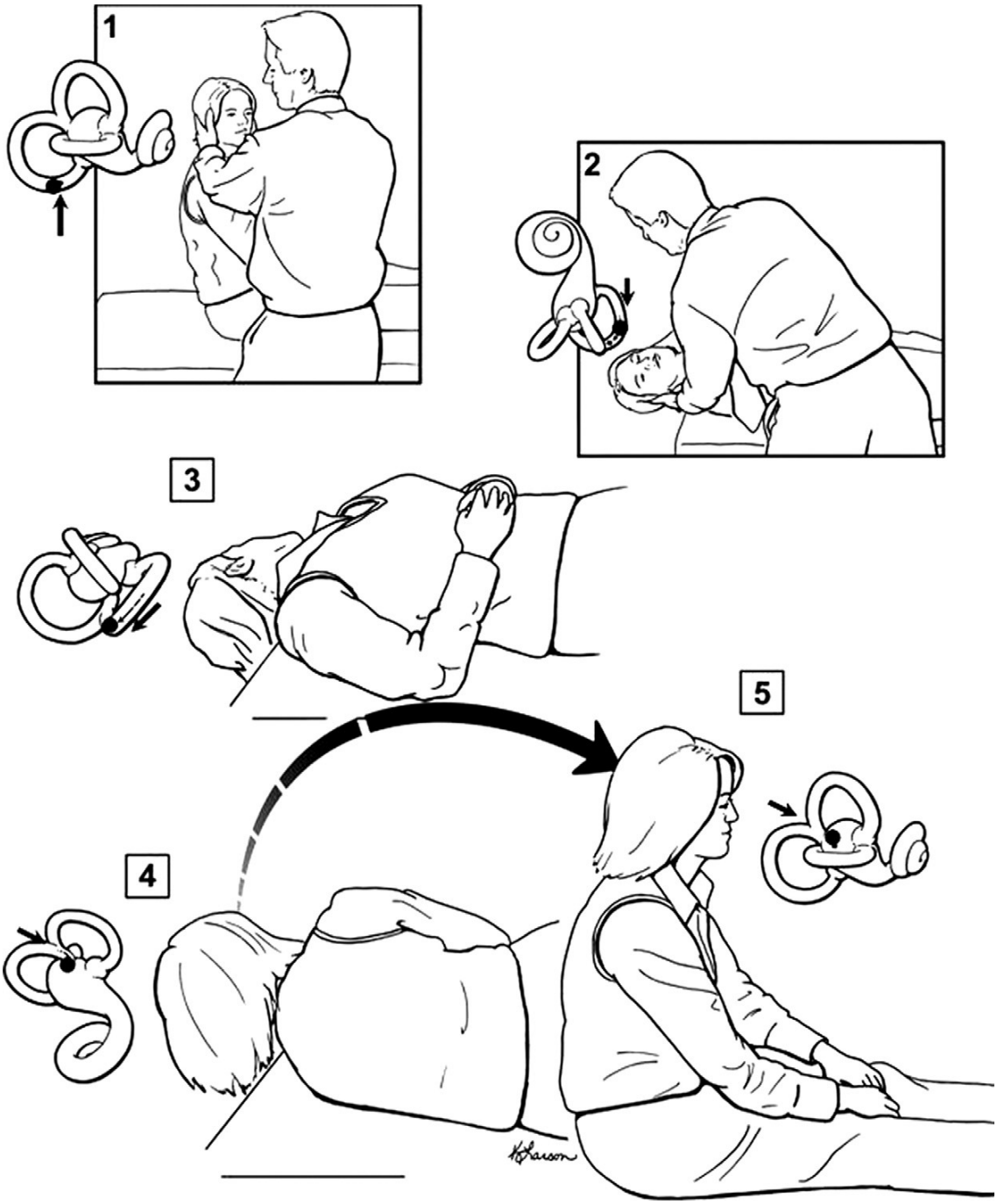

Figure 5.1 Performance of the therapeutic canalith repositioning procedure for right-sided posterior canal BPPV. (Adapted from reference (Fife, et al., 2008).) (**1**) The patient is placed in the upright position with the head turned 45 degrees toward the affected ear (the ear that was positive on the Dix-Hallpike testing). (**2**) The patient is rapidly laid back to the supine head-hanging position, which is then maintained for 20 to 30 seconds. (**3**) Next, the head is turned 90 degrees toward the other (unaffected) side and held for about 20 seconds. (**4**) Following this rotation, the head is turned a

further 90 degrees (usually necessitating the patient's body to also move from the supine position to the lateral decubitus position) such that the patient' head is nearly in the facedown position. This position is also held for 20 to 30 seconds. (**5**) The patient is then brought into the upright sitting position, completing the maneuver.

With respect to complications of treatment, CRP is associated with mild and generally self-limiting adverse effects in about 12 percent of those treated (Fife, et al., 2008). Serious complications from the CRP have not been identified in multiple RCTs. The most commonly encountered complications include nausea, vomiting, fainting, and conversion to lateral canal BPPV during the course of treatment (so-called canal switch). Such a canal switch occurs in about 6 to 7 percent of those treated with CRP, (Yimtae, et al., 2003) (Herdman, et al., 1996) underscoring the importance of recognizing the lateral canal variant of BPPV. Anecdotally, several investigators have suggested that the CRP should be applied cautiously in patients with cervical spine disease, certain vascular conditions, retinal detachment, and other contraindications to its performance (Sridhar, et al., 2005).

Table 5.1
Randomized controlled trials evaluating the effectiveness of CRP for posterior canal BPPV

Reference	Improved in treatment group n/N (%)	Improved in control group n/N (%)	Endpoint	Time to assessment	*P* value	Odds ratio (95% CI)	NNT
Lynn, 1995	11/18 (61%)	3/20 (15%)	Vertigo resolution	2 weeks	0.033	6.3 (1.29-30.5)	2.2
Froehling, 2000	12/24 (50%)	5/26 (19%)	Vertigo resolution	1-2 weeks	0.020	4.2 (1.2–14.8)	3.3
Simhadri, 2003	19/20 (95%)	3/20 (15%)	Vertigo resolution	1 week	0.001	107.7 (10.2-1135.5)	1.3
	19/20 (95%)	3/20 (15%)		4 weeks	0.001	107.7 (10.2-1135.5)	1.3
Waleem, 2008	16/22 (73%)	6/22 (27%)	Vertigo resolution	1 week	0.001	?	?
	18/22 (82%)	7/22 (32%)	Vertigo resolution	2 weeks	0.002	?	?
Yimtae, 2003	12/29 (41%)	1/27 (4%)	Vertigo resolution	1 week	0.005	18.4 (2.2-154.4)	2.7
	16/25 (64%)	7/20 (35%)		4 weeks	0.336	3.3 (1.0-11.3)	3.4
Cohen, 2005	*/24 (CRP)	*/25 (CRP)	Vertigo frequency scale (0-10)	4 weeks †	0.021		
	*/25 (LM)	*/25 (LM)		4 weeks †	0.010		
Von Brevern, 2006	28/35 (80%)	4/31 (13%)	Vertigo resolution	24 hours	0.001	27.0 (7.1-109.9)	1.5
Sugita, 2009	ROM 4/10 (40%)	CRP 6/12 (50%)	Vertigo resolution	1 week	No significant differences	NA	NA
	7/10 70%	9/12 75%		2 weeks			
	10/10 100%	9/12 75%		4 weeks			
Chang, 2008	CRP including vestibular stimulated exercise	CRP	Balance tests, Tandem walk test, Dynamic Gait index, Subjective	2 weeks	P<0.05	NA	NA
				4 weeks	P<0.05	NA	NA

	rating of the intensity of vertigo	

CI, confidence interval; *CRP,* canalith repositioning procedure; *LM,* Semont's liberatory maneuver; *NNT,* number needed to treat.
*Responses were analyzed with multilevel methods and expressed as fitted linear regression graphs, so no discrete numerical
expression of the response rates could be determined.
†Time to evaluation was varied, so data presented are based on fitted linear regression curves at 4 weeks.

Mastoid vibration. Mastoid vibration was included in the original Epley repositioning maneuver. One study, (Sridhar, et al., 2005) comparing patients with posterior canal BPPV treated by "appropriate canalith repositioning maneuvers," performed with and without vibration, showed no difference in immediate symptom resolution or relapse rate between groups.

Post-treatment activity restriction One study (Chang, et al., 2004) compared patients treated by CRP with and without mastoid vibration. There was no difference in symptom relief between the groups at 4 to 6 weeks (p 0.68). Two other studies (Lopez-Escamaez, et al., 1999) (Woodworth, et al., 2004) showed no difference in the rate of symptom resolution between patients treated by a CRP with or without mastoid vibration. Another study (Li, et al., 1995) reported that of patients treated by a CRP with vibration, 92% were "improved," vs 60% improvement with CRP alone.

In two studies (Lynn, et al., 1995; Froehling, et al., 2000) demonstrating the benefit of CRP, patients wore a cervical collar for 48 hours and avoided sleeping on the affected side for 1 week. Three studies (von Brevern, et al., 2006; Yimtae, et al., 2003; Cohen, et al., 2005) demonstrated the benefit of CRP and used no post-treatment restrictions or instructions. These studies were not designed to determine whether such restrictions affect treatment success; however, there seems to be little difference in the rate of treatment success whether or not restrictions were included. Six studies comparing CRP with and without post-treatment activity restriction were identified (Massoud, et al., 1996) (Blakley, et al., 1994) (Lempert, et al., 1997) (Wolf, et al., 1999) (Asawavichianginda, et al., 2009) (Angeli, et al., 2003). Five studies (Massoud, et al., 1996) (Blakley, et al., 1994) (Lempert, et al., 1997) (Wolf, et al., 1999) (Asawavichianginda, et al., 2009) showed no added benefit from post-treatment activity restriction or positions. Only one study showed a minimal benefit in patients with post-activity restrictions, as measured by the number of maneuvers required to produce a negative Dix–Hallpike maneuver (Asawavichianginda, et al., 2009).
Comparison of studies, in particular the treatment arms for RCTs, reveals similar response rates whether or not posttreatment positional or activity restrictions (ie, cervical collar or positional avoidance) are observed (Cohen, et al., 2005) (Froehling, et al., 2000) (Lynn, et al., 1995) (Yimtae, et al., 2003) (von Brevern, et al., 2006). Two studies looking at posttreatment restrictions after CRP found no evident improvement in those given restrictions (Massoud, et al., 1996) (Roberts, et al., 2005). Another study found slight benefit in patients with post-activity restrictions, as measured by the number of maneuvers required to produce a negative Dix-Hallpike maneuver (Cakir, et al., 2006). Overall, there is insufficient evidence to recommend post-maneuver restrictions in patients treated with CRP.

Treatment with the liberatory (Semont's) maneuver. Clinical trials concerning the treatment effectiveness of the liberatory maneuver (Fig 5.2) are limited. One study, (Cohen, et al., 2005) which included a treatment

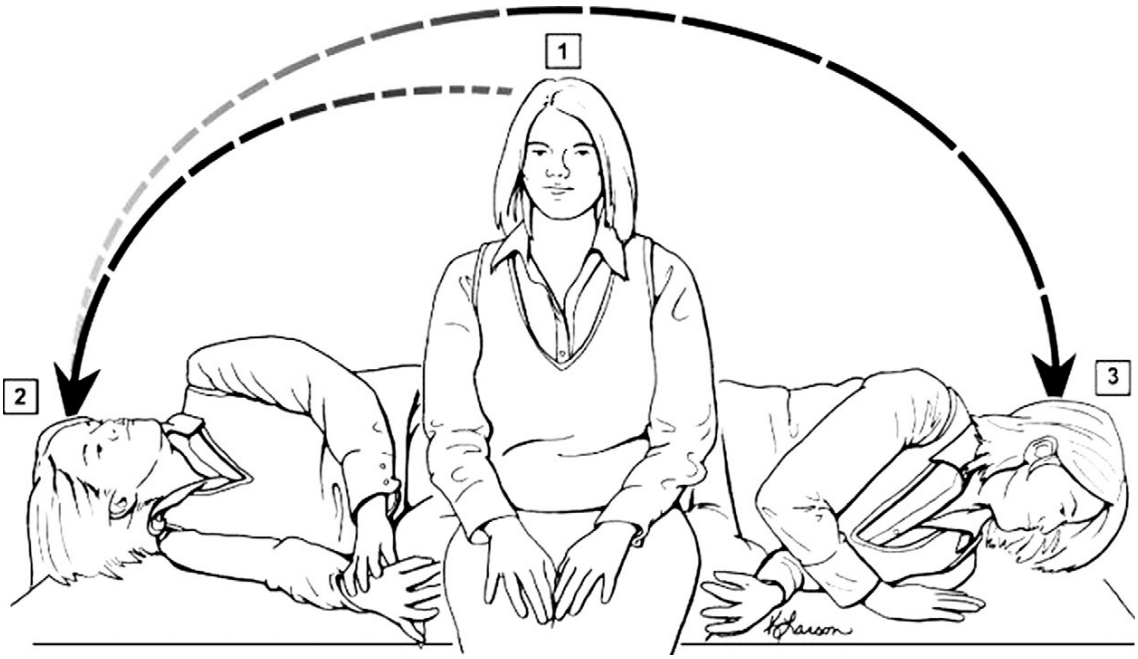

Figure 5.2 The Semont maneuver for right-sided BPPV. **(1**) Patient is seated in the upright position; then the patient's head is turned 45 degrees toward the left side, and the patient is then rapidly moved to the side-lying position as depicted in position (2). This position is held for approximately 30 seconds, and then the patient is rapidly moved to the opposite side-lying position without pausing in the sitting position and without changing the head position relative to the shoulder, resulting in position (3). This position is maintained for 30 seconds and then the patient gradually resumes the upright sitting position. (Adapted from reference (Fife, et al., 2008).)

arm with the Semont maneuver, demonstrated that this maneuver improved vertigo intensity more than the sham treatment (*P* _ 0.009). A study by Salvinelli et al (Salvinelli, et al., 2003) randomized 156 patients to the Semont maneuver, flunarizine (a calcium channel blocker), or no treatment. At 6-month follow-up, symptom resolution occurred in 94.2 percent of patients treated with the Semont maneuver, 57.7 percent of patients treated with flunarizine, and 34.6 percent of untreated patients. Soto Varela et al (Soto Varela, et al., 2001) randomized patients to treatment with CRP, Semont maneuver, or Brandt-Daroff exercises. Symptom resolution among those treated with either CRP or Semont maneuver at 1 week was the same (74% vs 71%) but only 24 percent for Brandt-Daroff exercises. At 3-month follow-up, however, patients treated with CRP demonstrated superior outcomes compared with those treated with Semont maneuver (*P* _ 0.027). In conclusion, the Semont maneuver is more effective than no treatment or Brandt-Daroff exercises in relieving symptoms of posterior canal BPPV, according to studies with small sample sizes and limitations. No adverse events have been reported in trials with the liberatory maneuver. Because of limited studies with direct comparisons between the liberatory maneuver and the CRP, no conclusions about differential effectiveness can be drawn.
Sugita proposed in a RCT that his rolling-over maneuver (ROM) is as effective as the canalith repositioning maneuver (CRP) for the treatment of posterior benign paroxysmal positional vertigo (BPPV). (Sugita, et al., 2009)

Lateral (Horizontal) Canal BPPV Treatments

Lateral canal BPPV is usually unresponsive to CRPs used for posterior canal BPPV but may respond to other maneuvers intended to move canaliths from the lateral canal into the vestibule (Herdman, et al., 1996) (Fife, et al., 2008) (Lempert, et al., 1996). The roll maneuver (Lempert maneuver or barbecue roll maneuver) or its variations are the most commonly employed maneuvers for the treatment of lateral canal BPPV (White, et al., 2005) (Prokopakis, et al., 2005). This maneuver involves rolling the patient 360 degrees in a series of steps to effect particle repositioning. It may be performed in the outpatient setting after a diagnosis of lateral canal BPPV has been made with the supine roll test.

Rather limited data exist with respect to the effectiveness of the roll maneuver in lateral canal BPPV treatment. Based primarily on cohort studies and case series, the effectiveness of the roll maneuver in treating lateral canal BPPV appears to be approximately 75 percent, although reported response rates vary widely from 50 percent to almost 100 percent (White, et al., 2005) (Fife, et al., 2008) (Nuti, et al., 1998) (Tirelli, et al., 2004) (Casani, et al., 2002) (Prokopakis, et al., 2005) (Fife, et al., 1998) (Lempert, et al., 1996) (Appiani, et al., 1997) (Sargent, et al., 2001) (Asprella Libonati, et al., 2005) (Chiou, et al., 2005). Because lateral canal BPPV may spontaneously remit more quickly than other forms of BPPV, a control group is especially important in assessing treatment efficacy (Moon, et al., 2006) (Sekine, et al., 2006).

Forced prolonged positioning is another treatment maneuver reported to be as effective in treatinglateral canal BPPV. It may be performed either alone or concurrently with other maneuvers with a reported effectiveness of 75-90 percent based on case series (Casani, et al., 2002) (Appiani, et al., 1997) (Chiou, et al., 2005) (Vannuchi, et al., 1997). Other lesser known maneuvers such as the Gufoni maneuver and the Vannucchi-Asprella liberatory maneuver (Asprella Libonati, et al., 2005) (Gufoni, et al., 1998) (Appiani, et al., 1997), have also been reported as effective in uncontrolled studies. In 2009 Francesco concluded that the Gufoni's manoeuvre is effective in treating patients suffering from BPPV of LSC; it is simple to perform; there are not many movements to execute, it needs low time of positioning, and positions are comfortable to the patient (Francesco, et al., 2009)

In conclusion, variations of the roll maneuver appear moderately effective and are the most widely used treatments for lateral canal BPPV. Other methods of treatment have also been advocated, but currently no RCTs provide reliable measures of effectiveness. At this time, there is insufficient evidence to recommend a preferred treatment maneuver for lateral canal BPPV treatment.

Anterior canal BPPV Treatment

The literature on the treatment of anterior canal BPPV is scarce. Two studies specifically looked into the treatment of anterior canal BPPV. One study showed that the canalith repositioning procedure is very effective in the treatment of anterior canal BPPV (CRP, also referred to as the Epley maneuver). One study treated 30 anterior canal BPPV patients with CRP. After on average 2 maneuvers 96.7% became symptom free (Kim et al., 2005). Another study suggests that the orientation of the ASC strongly facilitates the whole procedure in all cases. The repositioning manoeuvres seem to use respectively different routes in order to achieve the movement of otoconia toward the utricle (Korres, et al., 2010).

Self-Administration and Posttreatment Restrictions

Three studies have assessed patient self-treatment for BPPV. One study found slightly greater improvement in those patients given instructions for self-administered CRP at home after initial CRP

in the office (Sargent, et al., 2001). Self-administered CRP appeared to be more effective (64% improvement) than self-treatment with Brandt-Daroff exercises (23% improvement) (Radtke, et al., 1999). Another study reported 95 percent resolution of positional nystagmus 1 week after self-treatment with CRP compared with 58 percent in patients who selftreated using a modified Semont maneuver (*P* _ 0.001) (Radtke, et al., 2004). No comparison studies have been published from which to make recommendations regarding self-treatment vs clinician- administered treatment of BPPV. In motivated individuals, self-treatment of BPPV may be an option.

Conclusie 4a

Niveau 2	Bij zekere en atypische p-BPPD zijn de 'canalith repositiemanoeuvre' (CRM, ook wel Epley manoeuvre genoemd) en 2) de bevrijdingsmanoeuvre (ook wel Semontmanoeuvre genoemd) bewezen effectief. Voor andere behandelstrategieën is geen kwalitatief goed bewijs. Er is onvoldoende bewijs dat mastoidvibraties de effectiviteit van een repositiemanoeuvre verhogen bij p-BPPD.

Niveau 3	Er zijn aanwijzingen dat de 'log roll manoeuvre' of Lempert manoeuvre effectief is bij de behandeling van h-BPPD. Andere manoeuvres zijn mogelijk ook effectief. Er is onvoldoende bewijs dat mastoidvibraties de effectiviteit van een repositiemanoeuvre verhogen bij h-BPPD.

Niveau 4	Er is onvoldoende bekend over de effectiviteit van repositie manoeuvres bij de behandeling van a-BPPD.

Niveau 4	Er is geen bewijs dat postmanoeuvre beperkingen de effectiviteit van een repositie manoeuvre bij p-BPPD beïnvloeden. Bronnen: niveau D: (Massoud, et al., 1996) (Blakley, et al., 1994) (Lempert, et al., 1997) (Wolf, et al., 1999) (Asawavichianginda, et al., 2009) (Asawavichianginda, et al., 2009)

Overwegingen

- Voordeel: Snel verdwijnen van de symptomen
- Nadeel: opwekken van draaiduizeligheid door het uitvoeren van de repositiemanoeuvre,
- Kosten: laag
  Afweging: De voordelen wegen op tegen de nadelen. Er is echter ook een aanzienlijke kans op spontaan herstel.
- Waarde oordeel: grote waarde doordat symptomen onmiddellijk verdwijnen en ook gezien het gemak waarmee de CRP kan worden uitgevoerd.
- Rol van de voorkeur van de patiënt: matig

Aanbeveling 4a

Bij een zekere en waarschijnlijke p-BPPD is behandeling met een Epley- of Semontmanoeuvre geïndiceerd.

Er zijn aanwijzingen dat bij een h-BPPD behandeling met de log-roll manoeuvre of Lempert manoeuvre effectief is.

Er is onvoldoende bekend over de effectiviteit van repositiemanoeuvres bij de behandeling van a-BPPD.

Behandeling van BPPD dient door een arts of specifiek daartoe geschoolde paramedicus te geschieden.

Tabel 5.2: Treatment of the affected semicircular canal

Semicircular canal involvement	Type	Treatment
Right posterior canal	Canalolithiasis	Right CRM (modified Epley without vibration)
	Cupulolithiasis	Right Epley versus right Semont followed by right CRM
Left posterior canal	Canalolithiasis	Left CRM (modified Epley without vibration)
	Cupulolithiasis	Left Epley versus left Semont followed by left CRM
Right anterior canal	Canalolithiasis	Right CRM (modified Epley without vibration)
	Cupulolithiasis	Right Epley versus right Semont followed by right CRM
Left anterior canal	Canalolithiasis	Left CRM (modified Epley without vibration)
	Cupulolithiasis	Left Epley versus left Semont followed by left CRM
Right horizontal canal	Canalolithiasis	Right horizontal canal CRM (BBQ roll)
	Cupulolithiasis	Right modified Brandt-Daroff for horizontal canal followed by right horizontal canal CRM
Left horizontal canal	Canalolithiasis	Left horizontal canal CRM (BBQ roll)
	Cupulolithiasis	Left modified Brandt-Daroff for horizontal canal followed by left horizontal canal CRM

(Overgenomen uit: Anterior canal beniging paroxysmal positional vertigo, Jackson et al, 2007, die hebben het aangepast en overgenomen uit Herdman SJ Advances in the treatment of vastibular disorders. Phys Ther 1997; 77: 602-18)

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Rosalia, Gargano [corrected to Gargano, Rosalia]; Riccardo, Speciale [corrected to Speciale, Riccardo]] (2009). *American Journal of Otolaryngology Mar; 30(2),* 106-11.

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Uitgangsvraag 4b:

Is vestibulaire revalidatie geschikt als therapie om patiënten met BPPD te behandelen?

Onderbouwing

The clinician may offer vestibular rehabilitation, either self-administered or with a clinician, for the initial treatment of BPPV. Option based on controlled observational studies and a balance of benefit and harm. Overview of Vestibular Therapy Vestibular rehabilitation is a form of physical therapy designed to promote habituation, adaptation, and compensation for deficits related to a wide variety of balance disorders. It may also be referred to as vestibular habituation, vestibular exercises, or vestibular therapy. There is no single specific protocol for vestibular rehabilitation, but rather a program of therapy is developed on the basis of the underlying diagnosis. Programs can include canalith repositioning exercises, adaptation exercises for gaze stabilization, habituation exercises, substitution training for visual or somatosensory input, postural control exercises, fall prevention training, relaxation training, conditioning exercises, functional skills retraining, and patient and family education (Herdman, et al., 2000) (Telian, et al., 1996) (Whitney, et al., 2000).

With respect to BPPV, vestibular rehabilitation programs most commonly focus on habituation exercises either in formal outpatient therapy programs or with home exercise programs. Vestibular rehabilitation programs may also include PRMs, but repositioning maneuvers will be covered separately in the guideline. Herein, we refer to vestibular rehabilitation as a series of exercises or training maneuvers performed by the patient for the treatment of BPPV with or without direct clinician supervision.

Vestibular rehabilitation habituation exercises were first described by Cawthorne and Cooksey in the 1940s (Cawthorne, et al., 1944). These exercises consist of a series of eye, head, and body movements in a hierarchy of increasing difficulty, which provokes vestibular symptoms. The exercises begin with simple head movements, performed in the sitting or supine position, and progress to complex activities, including walking on slopes and steps with eyes open and closed, and sports activities requiring eye-hand coordination. These exercises theoretically fatigue the vestibular response and force the CNS to compensate by habituation to the stimulus (Norre, et al., 1987a/b). In 1980, Brandt and Daroff (Brandt, et al., 1980) (Brandt, et al., 1994) described home repositioning exercises that involve a sequence of rapid lateral head/trunk tilts repeated serially to promote loosening and ultimately dispersion of debris toward the utricular cavity. In these exercises, the patient starts in a sitting position and moves quickly to the right-side lying position, with the head rotated 45 degrees and facing upward. This position is maintained for 30 seconds after the vertigo stops. The patient then moves rapidly to a left-side lying position, with the head rotated 45 degrees and facing upward. In early work with patients with BPPV, patients repeated these maneuvers moving from the sitting to side-lying position three times a day for 2 weeks while hospitalized and had excellent resolution of BPPV symptoms (Troost, et al., 1992).

<u>Vestibular Rehabilitation as a Treatment of BPPV</u>

Relatively few RCTs and case series have been published regarding the effectiveness of vestibular rehabilitation as the initial therapy for BPPV. In a prospective analysis of 25 consecutive patients with BPPV, Banfield et al (Banfield, et al., 2000) reported that patients demonstrate an excellent short-term response rate of 96 percent subjectively to vestibular rehabilitation treatment with an average of three clinic visits per patient, but the authors noted a significant recurrence rate of BPPV with long-term follow-up (mean follow-up 3.8 years). The authors cited one advantage of vestibular rehabilitation: the capability of patients to be self-reliant in their ability to return to habituation exercises should symptoms recur. In a controlled trial of 60 patients with BPPV comparing a PRM,

vestibular rehabilitation exercises and no treatment, vestibular rehabilitation provided better resolution of vertigo compared with no treatment (Steenerson, et al., 1996). The PRM arm demonstrated resolution of symptoms with fewer treatments than those required for vestibular rehabilitation, although the relative improvements at 3-month follow-up were comparable.

Several studies have compared vestibular rehabilitation exercises to particle rehabilitation maneuvers in the treatment of posterior canal BPPV. In an RCT of 124 patients randomized to CRP, modified liberatory maneuver, sham maneuver, Brandt-Daroff exercises, and vestibular habituation exercises by Cohen, repositioning maneuvers were more effective than Brandt-Daroff exercises or habituation exercises (Cohen et.al., 2005). Both types of vestibular rehabilitation treatments, however, were individually more effective than a sham intervention (Cohen, et al., 2005)(Hillier, 2007). Soto Varela et al (Soto Varela, et al., 2001) comparatively analyzed a total of 106 BPPV patients randomly assigned to receive Brandt-Daroff habituation exercises, the Semont maneuver, or the Epley maneuver. At the 1-week follow-up, similar cure rates were obtained with the Semont and Epley maneuvers (74% and 71%, respectively), both cure rates being significantly higher than that obtained with Brandt- Daroff exercises (24%). At 3-month follow-up, the cure rate for the Brandt-Daroff exercises increased significantly to 62 percent, although the rate was still lower than that of PRMs. Other studies have demonstrated similar results for vestibular rehabilitation in BPPV (Furman, 1999) (Toledo, 2000). In a double blind RCT control study Chang WC et al. demonstrated that additional exercise training, which emphasizes vestibular stimulation, can improve balance ability and functional gait performance among patients with benign paroxysmal positional vertigo of the posterior semicircular canal who had already undergone the canalith repositioning manoeuvre (Chang, et al., 2008).

Vestibular rehabilitation is thought to improve long-term outcomes for BPPV. Although data are mixed, a few studies have indicated that use of vestibular rehabilitation may decrease recurrence rates for BPPV (Angeli, et al., 2003) (Helminski, et al., 2005). This protective effect against recurrence of vestibular rehabilitation may be more pronounced in the elderly (Angeli, et al., 2003). Several prospective studies have demonstrated the safety and effectiveness of vestibular rehabilitation for unilateral peripheral vestibular disorders; the results are summarized in a recent Cochrane collaboration report (Hillier, et al., 2007). Among 21 included randomized trials, there were no reports of adverse effects due to vestibular rehabilitation therapy. Current published evidence is inadequate to indicate superiority for one form of vestibular rehabilitation vs another. There is also not enough evidence to favor formal outpatient vestibular therapy performed with a clinician over independent home therapy (Kammerlind, et al., 2005).

In summary, with respect to posterior canal BPPV, vestibular rehabilitation demonstrates superior treatment outcomes compared with placebo. In short-term evaluation, vestibular rehabilitation is less effective at producing complete symptom resolution than PRMs. With longer-term follow-up, however, its effectiveness approaches that of PRMs. Insufficient data exist concerning the response of lateral canal BPPV to vestibular therapy; this area needs further research.

Cost considerations may become important if repeated visits for clinician-supervised therapy are required as opposed to initial patient instruction followed by home-based therapy. Patients with certain comorbidities may not be appropriate candidates for vestibular rehabilitation or may need specialized, individually tailored vestibular rehabilitation protocols. Examples of such comorbidities include cervical stenosis, Down syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, and spinal cord

injuries. On the other hand, patients with preexisting otological or neurological disorders may derive more benefit from vestibular rehabilitation as a treatment for BPPV.

Niveau 2/3	Vestibulaire revalidatie in de vorm van adaptatieoefeningen is bij posterieure kanaal BPPD effectief in vergelijking met placebo. Op korte termijn lijkt vestibulaire revalidatie minder effectief dan een repositiemanoeuvre, maar op lange termijn is vestibulaire revalidatie mogelijk net zo effectief als een repositiemanoeuvre. Er zijn aanwijzingen dat, vooral bij ouderen, vestibulaire revalidatie als aanvullende therapie naast een repositiemanoeuvre beter zou beschermen tegen het terugkeren van de klachten dan een repositiemanoeuvre alleen. Er zijn onvoldoende gegevens om de effectiviteit van vestibulaire therapie bij horizontale kanaal BPPD te beoordelen.

Overwegingen

- Voordeel: potentieel sneller verlost van symptomen dan met observatie alleen.
- Nadeel: geen ernstige bijwerkingen gezien in de gepubliceerde trials, provocatie van BPPD symptomen door revalidatie oefeningen, mogelijk een tragere oplossing dan repositie manoeuvres.
- Kosten: noodzaak tot herhaalde bezoeken
- Afwegingen: de voordelen wegen niet op tegen de nadelen, wel kan bij ouderen vestibulaire revalidatie worden overwogen, als aanvullende therapie na een repositiemanoeuvre.
- Waarde oordeel: vestibulaire revalidatie is mogelijk beter geschikt als aanvullende therapie dan als primaire behandeling (een subgroep van patiënten met balansstoornissen, centraal zenuwstelsel stoornissen of risico om te vallen, kunnen meer profiteren van vestibulaire revalidatie dan patiënten met uitsluitend BPPD).
- Rol van de voorkeur van de patiënt: aanzienlijk gezien de gezamenlijke beslissing
- Exclusiecriteria: patiënten met fysieke beperkingen

Uitgangsvraag 4c:

Zijn medicijnen geschikt als therapie om patiënten met BPPD te behandelen?

The symptoms of vertigo due to many different underlying etiologies are commonly treated with medications. Clinicians may prescribe pharmacological management to either 1) reduce the spinning sensations of vertigo specifically and/or 2) to reduce the accompanying motion sickness symptoms. These motion sickness symptoms include a constellation of autonomic or vegetative symptoms such as nausea, vomiting, and diarrhea, which can accompany the vertigo. Such pharmacological therapies for vertigo may be broadly termed *vestibular suppressant medications* (Hain, et al., 2003) (Hain, et al., 2005). Several categories of vestibular suppressant medications are in common use. Of these, the most commonly used are benzodiazepines and antihistamines. Benzodiazepines, such as diazepam and clonazepam, have anxiolytic, sedative, muscle relaxant, and anticonvulsant properties derived from potentiating the inhibitory effect of the gamma-amino butyric acid system. In prolonged dizziness, these medications can reduce the subjective sensation of spinning, but they also interfere with central compensation in peripheral vestibular conditions. Antihistamines, on the other hand, appear to have a suppressive effect on the central emetic center to relieve the nausea and vomiting associated with motion sickness. Common examples of antihistamines used to treat symptoms of

vertigo and/or associated motion sickness include meclizine and diphenhydramine. Other medications that are often used for motion sickness include promethazine, which is a phenothiazine with antihistamine properties, and ondansetron, which is a serotonin-5-hydroxytryptamine- 3 antagonist. Finally, anticholinergic medications such as scopolamine block acetylcholine, which is a widespread CNS transmitter, and help with motion sickness by reducing neural mismatching (Hain, et al.,., 2003) (Hain, et al., 2005).

There is no evidence in the literature to suggest that any of these vestibular suppressant medications are effective as a definitive, primary treatment for BPPD, or as a substitute for repositioning maneuvers (Frohman, et al., 2003) (Hain, et al., 2003) (Carlow, et al., 1986) (Cesarani, et al., 2004) (Fujino, et al., 1994). Exercise was found to be a better treatment choice than medication (betahistin) and may be preferable for patients with persistent or chronic vertigo (Kulcu, et al., 2008).Some studies show a resolution of BPPD over time with medications, but these studies follow patients for the period of time in which spontaneous resolution would occur (Woodworth, et al., 2004) (Salvinelli, et al., 2004) (Itaya, et al., 1997) (McClure, et al., 1980). In one double- blind controlled trial by McClure and Willet (McClure, et al., 1980) comparing diazepam, lorazepam, and placebo, all groups showed a gradual decline in symptoms with no additional relief in the drug treatment arms. In a small study, Itaya et al (Itaya, et al., 1997) compared PRMs to a medication-alone treatment arm and found that PRMs had substantially higher treatment responses (78.6%-93.3% improvement) compared with medication alone (30.8% improvement) at 2 weeks follow-up. These data reinforced previous data from Fujino et al (Fujino, et al., 1994) that also indicated superiority of vestibular training for BPPD over medication use alone. A lack of benefit from vestibular suppressants and their inferiority to PRMs indicate that clinicians should not substitute pharmacological treatment of symptoms associated with BPPD in lieu of other more effective treatment modalities.

Conversely, vestibular suppressant medications have the potential for significant harm. All of these medications may produce drowsiness, cognitive deficits, and interference with driving vehicles or operating machinery (Ancelin, et al., 2006) (Hebert, et al., 2007) (Barbone, et al., 1998) (Engeland, et al., 2007) (Jauregui, et al., 2006). Medications used for vestibular suppression, especially psychotropic medications such as benzodiazepines, are a significant independent risk factor for falls (Hartikainen, et al., 2007). The risk of falls increases in patients taking multiple medications and with the use of medications such as antidepressants (Lawson, et al., 2005) (Hien, et al., 2005). The potential for polypharmacy when adding vestibular suppressants further exposes the elderly to additional risk (Landi, et al., 2007). Educational programs to modify practitioner's use of such medications can result in a reduction of falls (Pit, et al., 2007).

There are other potential harmful side effects of vestibular suppressants. Benzodiazepines and antihistamines interfere with central compensation for a vestibular injury (Hanley, et al., 1998) [Baloh, 1998] (Baloh, et al., 1998). The use of vestibular suppressants may obscure the findings on the Dix-Hallpike maneuvers. In addition, there is evidence of additional potential harm from the antihistamine class of medications on cognitive functioning (Ancelin, et al., 2006), and on gastrointestinal motility, urinary retention, vision, and dry mouth in the elderly (Rudolph, et al., 2008).

Another type of medication, betahistine dihydrochloride is an extensively applied and studied drug in the treatment of vertigo as well, especially in case of Meniere Disease. Betahistine appears to be a weak H1 agonist (release of Ca2++), a weak H2 agonist (synthesis cyclic AMP) and a strong H3 antagonist (release of histamine)(Timmerman, et al., 1989). The action upon the H3 auto-receptor might explain why a relatively low concentration of betahistine could effectively modify

neurotransmission in the brain (Tighilet, et al., 1995). Betahistine also results in a dose-dependent inhibition of polysynaptic neurons in the lateral vestibular nuclei (KawabataA). Recently it was shown that betahistine fastens the central compensation process after labyrinthectomy in cats (Tighilet, et al., 1995) and after neurectomy in men (Redon et al., 2010), In animals intra-labyrinthine blood flow and oxygenation of sensory tissue is increased by intake of betahistine (Meyer et al., 1994, Laurikainen et al., 1993). In one study, the treatment of patients with BPPV was found not to be effective in BPPD (Kulcu, et al., 2008).

In summary, vestibular medications are not recommended for treatment of BPPD, other than for the short-term management of vegetative symptoms such as nausea or vomiting in a severely symptomatic patient. Examples of potential short-term uses include patients who are severely symptomatic yet refuse therapy or patients who become severely symptomatic after a PRM. Antiemetics may also be considered for prophylaxis for patients who have previously manifested severe nausea and/or vomiting with the Dix-Hallpike maneuvers and in whom a PRM is planned. If prescribed for these very specific indications, clinicians should also provide counseling that the rates of cognitive dysfunction, falls, drug interactions, and machinery and driving accidents increase with use of vestibular suppressants.

Conclusie

Niveau 3	Medicatie is niet effectief als behandeling van BPPD.

Overwegingen

- Voordeel: patiënt krijgt geen onterechte medicatie
- Nadeel: geen
- Kosten: voordelig om geen medicatie voor te schrijven
- Afweging: voordelen wegen op tegen de nadelen. Kortdurend gebruik van vestibulosuppressieve medicatie en/of anti-emetica kan soms wel zinvol zijn om een Dix-Hallpikemanoeuvre of repositiemanoeuvre te kunnen uitvoeren teneinde misselijkheid en/of braken te voorkomen.
- Waarde oordeel: Schade door ineffectieve behandeling wordt voorkomen.
- Rol van de voorkeur van de patiënt: is minimaal.
- Exclusiecriteria: Patiënten die profylaxe voor Dix-Hallpikemanoeuvre en/of repositie manoeuvre nodig hebben.

Aanbeveling

Er is geen indicatie om patiënten met BPPD medicatie voor te schrijven.

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Uitgangsvraag 4d:

Is chirurgische interventie geschikt als behandeling?

Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common disorders in patients suffering from vertigo. The pathogenesis is currently thought to be canalolithiasis or cupulolithiasis of the posterior semicircular canal (Epley, et al.,1990). Canalith repositioning procedures by which densities are removed from the responsible canal are widely accepted in the treatment of patients with BPPV (Epley, et al.,1992; Seo et.al., 2007). Positional vertigo is immediately resolved by the treatment; however, a few patients continue to complain of vertigo after treatment (Seo et al., 2007).

According to the canalolithiasis theory, the symptoms should improve when the endolymphatic current of the responsible canal is blocked. Parnes and McClure were the first to report that positional vertigo completely disappeared after posterior semicircular canal occlusion surgery in two cases of intractable BPPV in 1990 (Parnes, McClure, et al., 1990 4). Although later studies also showed that plugging surgery has a high success rate for resolving positional vertigo and positional nystagmus, it is not widely performed. One reason is a risk of inner ear injury that causes severe sensorineural hearing loss (SNHL) and prolonged disequilibrium.

Samenvatting van de literatuur

Canal plugging

One evidence-based review was found that adressed the question whether surgical occlusion of the posterior canal or singular neurectomy is effective for BPPV (Fife et.al., 2008). The most common used procedure in this studies is fenestration and occlusion of the posterior semicircular canal. Five studies of level D, including in total 86 patients undergoing canal occlusion, reported 'complete relief' of BPPV symptoms in 85 as ascertained by the treating surgeon. Reported complications included a 'mild' conductive hearing loss for 4 weeks or less, 'mild' and transient' unsteadiness in most patients, and a high frequency sensorineural hearing loss in 6 patients.

Two other level D studies were found that used canal plugging to treat BPPV patients. One study described results of 48 patients. A limitation of this study was that inclusion criteria for surgery were not clearly described. It was not clear whether these patients suffered from intractable BPPV. Out of 44 patients with normal preoperative hearing, one had a delayed (3-months) sudden and permanent profound loss, while another had a mild (20dB) loss. Six patients had protracted courses of imbalance and motion sensitivity (Agrawal and Parnes, et al., 2005). The other study specifically looked into the effect of posterior semicircular canal plugging surgery on hearing and balance functions. The patients included in this study were five consecutive patients with intractable BPPV, 1% of all BPPV patients treated in this period, who underwent plugging surgery. After surgery positional vertigo was resolved in all patients. This study showed that results of audiometry, caloric testing and vestibular evoked myogenic potential (VEMP) testing were hardly influenced by plugging surgery (Seo et.al., 2009).

Singular neurectomy

Singular neurectomy is another surgical treatment for intractable BPPV. It relieves the positional vertigo by deafferentiating the ampulla of the posterior semicircular canal. In this procedure, the singular canal in the round window niche is identified and the nerve transected. In one study 96.8% were reported to have 'complete relief' of vertigo after singular neurectomy (Gacek, et al., 1974).
Further reports by Gacek demonstrated high efficacy with complete vertigo resolution in 91.7% of patients (Gacek, et al., 1978). A non-systematic review (Leveque et al., 2007) included five other studies that performed singular neurectomy. Complete relief was seen in 75% to 96% of cases according to the test performed by the surgeon. Partial relief occurred in 1.5% to 17% of patients, which is defined by reduction but not absence of vertigo and nystagmus triggered by a provocative maneuver. There were some patients whose symptoms did not improve after surgery, mostly because of incorrect diagnoses, failure to locate the singular nerve, and incomplete transsection of the posterior ampullary nerve due to the presence of an accessory branch. Another study, not included in this review, evaluated eight patients with BPPV, one with typical symptoms, but without nystagmus at the Hallpike's manoeuvre (Pournaras et al., 2008). After singular neurectomy all patients were free of vertigo, but sensorineural hearing loss occurred in two patients.

Sensorineural hearing loss depended on the surgeon's experience. Gacek et al. reported an initial 7.3% risk of sensorineural hearing loss with the procedure. This risk was decreased in an update of his personal series in 1995 to 3%, for 252 neurectomies (Gacek 1995; Agrawal et al., 2005). Other reported a risk of hearing loss that ranged from 9% (Silverstein, et al., 1990), 19% (Meyerhoff, et al., 1985) to 41% of cases for Epley (12 neurectomies) (Epley, et al., 1980). It ranges from a 30 dB hearing loss to total deafness. Most of those sensorineural hearing losses can be explained by the surgical act (injury to the scala tympani, to the perilymphatic space or, worse, to the endolymphatic space). Sensorineural hearing losses that remain unexplained may be due to secondary labyrinthitis.

Patients experienced dizziness for a few days after the intervention; for that period, a vertical downbeat nystagmus can be seen that is known to be of cerebellar origin. Silverstein and White explored the vestibular function on operated patients. They found that 41% of them had vestibular dysfunction with no clinical consequences whereas there were only 14% before operation (Leveque et al., 2007).

Comparison with canal occlusion:

Although singular neurectomy seems safe in experienced hands, it is technically very demanding with a high risk of sensorineural hearing loss (Schessel, et al., 1998). This may explain why it has largely been replaced by the simpler semicircular canal occlusion.

Conclusies

Niveau 3	Het lijkt waarschijnlijk dat canal plugging een effectieve en veilige behandelmethode voor BPPD is als herhaalde repositiemanoeuvres niet werken. *bronnen niveau D: Fife et.al., 2008, Agrawal and Parnes, et al., 2005, Seo et.al., 2009*

Niveau 3/4	Er zijn aanwijzingen dat neurectomie van de n. singularis effectief is bij de behandeling van BPPD, maar deze ingreep gaat gepaard met een grote kans op bijwerkingen zoals perceptief gehoorsverlies. *bronnen niveau D: (Gacek et al.,),* (Schessel, et al., 1998)*, (Leveque, et al., 2007)*

Overwegingen

- Voordeel: bewezen effectiviteit
- Nadelen: kans op perceptieslechthorendheid, kans op (blijvende) evenwichtsstoornissen. Neurectomie is een technisch veeleisende operatie waarbij de ervaring van de chirurg een grote rol speelt.
- Kosten: fors
- Afweging van voordeel tegen nadeel: bij therapieresistente BPPD moeten de risico's van een operatieve ingreep worden afgewogen tegen de ernst van de klachten/invalidering door de klachten
- Waarde oordelen:
- Rol van de voorkeur van de patiënt: groot
- Exclusie: geen

Aanbevelingen

De werkgroep beveelt aan om bij ernstig invaliderende, therapieresistente, zekere BPPD canal plugging te overwegen. De kans op ernstige bijwerkingen zoals doofheid en blijvende evenwichtsstoornissen dient goed met de patiënt te worden besproken.
Neurectomie van de n. singularis is een techniek die gezien de complexiteit voorbehouden is aan gespecialiseerde KNO-artsen.

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Uitgangsvraag4e:

Kan bij patiënten met BPPD volstaan worden met een expectatief beleid?

Observation may be defined as a “watchful waiting” or the withholding of specific therapeutic interventions for a given disease. Observation is often considered when the disease course is self-limited and/or felt to be benign with limited sequelae occurring from the withholding of therapy. In BPPV, observation implies that therapeutic interventions such as vestibular rehabilitation and/or PRMs will be withheld, anticipating a natural and spontaneous improvement of the symptoms of BPPV. Under a course of observation, patients may still be instructed to avoid provocative positions and activities where the risk of injury (ie, falls) may be increased until symptoms resolve spontaneously or until they are reassessed for symptom resolution.

To consider observation as an option in the management of BPPV, the clinician must determine the natural history of the BPPV. It has been presumed that the natural history of BPPV is one of eventual resolution in most patients. It should be noted, however, that an often quoted study by Blakley, (Blakley, et al., 1994) which reported high rates of spontaneous resolution of BPPV, relied on subjective symptom reporting, rather than objective testing with a Dix-Hallpike maneuver, as the outcome measure for resolution. It is believed that a significant fraction of patients reporting subjective improvement actually have reduction in symptoms secondary to avoiding provocative (vertigo-producing) positions rather than actual cure (Woodworth, et al., 2004). More recent RCTs have utilized objective testing with the Dix-Hallpike maneuver as an additional outcome measure to assess for objective resolution of BPPV. Notably, to observe proper blinding, most RCTs also use a sham positional maneuver in the control group, which theoretically may affect the natural history of BPPV.

In several studies, the spontaneous rate of symptomatic resolution of BPPV ranges from 15 to 86 percent. The reported rate of spontaneous improvement based on objective positional testing (ie, conversion to a negative Dix- Hallpike maneuver) ranges from 35 percent to 50 percent (Woodworth, et al., 2004). As demonstrated in Table 5.1, the natural history of posterior canal BPPV varies widely across studies at a 1-month and a 3-month follow-up interval. Further variability in the spontaneous resolution rate arises from differences in duration of symptoms prior to actual diagnoses of BPPV as well as differences in duration of follow-up (Hilton, et al., 2004) (Froehling, et al., 2000) (Lynn, et al., 1995) (Sekine, et al., 2006). Longitudinal follow-up studies of untreated BPPV patients are lacking, but one study of completely untreated patients determined a mean time interval from onset of symptoms to spontaneous resolution of BPPV of 39 _ 47 days (Imai, et al., 2005). As would be expected, spontaneous symptom resolution rates increase with increasing duration of follow-up among observed patients.

Tabel 5.1: Symptoom reductie rates voor observatie alleen van BPPD

Referentie	Genezen n/m	% genezen	Placebo behandeling of pure observatie	Tijd tot assessment
Von Brevern, 2007	22/26	84.6%	Placebo	4 weken
Sakine 2006	48/60	80.0%	Observatie	1 maand
Imai 2005	45/70	64.0	Observatie	1 maand
Simhadri 2003	3/15	20	Observatie	4 weken
Yimtae 2003	7/20	35.0	Observatie	1 maand
Sherman 2001	11/22	50.0	Placebo	3 maanden
Asawavichianginda 2000	18/22	81.8	Observatie	3 maanden
Steenerson 1996	17/40	42.5	Observatie	3 maanden
Lynn 1995	3/15	20.0	Placebo	1 maand
Blakley 1994	19/22	86.4	Observatie	1 maand

Eindpunt: herstel van vertigo symptomen op het tijdstip van assessment.

Although observation of posterior canal BPPV is an option for management, clinicians should also be aware that other treatments such as the PRM have been shown to offer patients faster resolution of BPPV symptoms. A meta-analysis of nine separate trials examining the efficacy of the PRM for BPPV treatment demonstrated consistent improvement in the treatment group, with up to 4.1 times greater rates of symptom resolution (95% confidence interval, 3.1- 5.2) in the PRM groups vs the control groups at initial assessments within 1 month. Studies with follow-up at beyond 1 month still demonstrated an improvement rate of nearly three times that of controls (Woodworth, et al., 2004). Other longer-term follow-up data also suggest that patients treated with a PRM had lower rates of relapse of BPPV at 6 months and 1 year posttreatment (Simhadri, et al., 2003).

Observation as an option for the management of posterior canal BPPV offers the potential benefits of avoiding repositioning maneuvers or vestibular rehabilitation, which in turn may provoke symptoms and discomfort. There may also be a cost savings from decreased rates of referral for vestibular rehabilitation or PRMs. From a potential harms perspective, patients who elect for the observation option should be informed about a typically longer duration of symptoms compared with a treatment maneuver and potentially higher recurrence rates. Appropriate precautions for the risks associated with BPPV symptoms should be taken during the watchful waiting period.

The natural history of lateral canal BPPV is less well defined than that of posterior canal BPPV. Several authors have commented that lateral canal BPPV may be prone to more rapid spontaneous resolution than posterior canal BPPV (Moon, et al., 2006) (Sekine, et al., 2006). In one study, the mean time between the onset of vertigo in lateral canal BPPV to spontaneous resolution was 16 _ 19 days (Imai, et al., 2005). Although repositioning maneuvers have shown success in lateral canal BPPV, overall high quality comparative data regarding treatment vs observation such as RCTs are limited in this subtype of BPPV (Casani, et al., 2002) (Sekine, et al., 2006) (Fife, et al., 2006). Thus, observation of lateral canal BPPV remains an option for management. Future RCTs need to be dedicated to the interventional management of lateral canal BPPV.

Niveau 2	De symptomen verdwijnen bij 35-50 procent van de patiënten na ongeveer 1 maand zonder interventie.

Overwegingen

- Voordeel: De symptomen verdwijnen bij 35-50 procent van de patiënten na ongeveer 1 maand zonder interventie.
- Nadeel: Langer last van symptomen dan bij behandeling waardoor de patiënt een groter valrisico heeft of niet in staat is tot werken.
- Kosten: indirect kosten vanwege een vertraagde genezing in vergelijking met andere behandelingen.
- Afweging: Een afwachtend beleid als behandeling voor posterieur kanaal BPPD heeft als voordeel dat de patiënt geen manoeuvres of vestibulaire revalidatie hoeft te ondergaan. Niet behandelen heeft echter als nadeel dat de klachten langer persisteren met mogelijke gevolgen voor kwaliteit van leven, vallen en arbeidsverzuim..
- Waarde oordeel: de werkgroep heeft een voorkeur voor een behandelingsinterventie in plaats van het natuurlijk beloop af te wachten, vooral omdat de klachten dan sneller verdwijnen.
- Rol van de voorkeur van de patiënt: substantieel gezien de gezamenlijke beslissing over de behandeling.
- Exclusiecriteria: geen

Aanbeveling

Behandelen van BPPD verdient de voorkeur boven het afwachten van het natuurlijk beloop.

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Hoofdstuk 6 Omgevingsfactoren BPPD

Uitgangsvraag 5:

Met welke factoren moet rekening worden gehouden bij de behandeling van BPPD?

Although BPPV arises from dysfunction of the vestibular end organ, patients with BPPV often concurrently suffer from comorbidities, limitations, and risks that may affect the diagnosis and treatment outcome of BPPV. Assessment of the patient with BPPV for factors that modify management is essential for improved treatment outcomes and ensuring patient safety with an underlying diagnosis of BPPV. The majority of factors that may modify management of BPPV can be identified if the clinician questions patients for these factors and elicits a detailed history (Rubenstein, et al., 2001).
Given that BPPV occurs most commonly in the second half of the lifespan and its prevalence increases with age, patients suffering from BPPV often have medical comorbidities that may alter the management of BPPV (Lawson, et al., 2005). In crossectional surveys, patients with BPPV demonstrate higher rates of diabetes, history of head trauma, and anxiety (Cohen, et al., 2004). Other studies have also found higher relative rates of migraine (34% in BPPV patients vs 10% in non-dizziness control group), history of stroke (10% in BPPV patients vs 1% in controls), diabetes (14% vs 5%), and hypertension (52% vs 22%) (von Brevern, et al., 2007). Clinicians should assess patients with BPPV for these comorbidities because their presence may modify management and influence treatment outcomes in BPPV.

One of the major concerns with BPPV and vertiginous syndromes in general is the risk for falls and resultant injury (Gazzola, et al., 2006). In multiple studies concerning etiology of falls, dizziness and vertigo were deemed the primary etiology for 13 percent of falls, compared with existing balance and gait problems (17%) and person-environment interactions (31%) (Rubenstein, et al., 2006). In a study by Oghalai,15 9 percent of patients referred to a geriatric clinic for general geriatric evaluation had undiagnosed BPPV, and three-fourths of those with BPPV had fallen within the 3 months prior to referral. Thus, evaluation of patients with a diagnosis of BPPV should also include an assessment of risk for falls (Lawson, et al., 2005). In particular, elderly patients will be more statistically at risk for falls with BPPV. Clinicians may use various fall assessment tools to determine the patient's fall risk and appropriate precautionary recommendations (Rubenstein, et al., 2001).

As noted above, comorbid conditions that occur commonly with BPPV such as a history of stroke or diabetes should also be identified during evaluation of patients with BPPV. Patients with a history of stroke or a history of diabetes, particularly with peripheral neuropathy, may already have preexisting gait, balance, or proprioceptive deficit (Casellini, et al., 2007) (Richardson, et al., 2002) (Tilling, et al., 2006). The additional symptoms of BPPV may increase their risk for fall and injury. Patients with visual disturbances often lack the ability to correct for or compensate for a balance deficit with visual cues, and may also be at increased risk for falls. Associations between osteopenia and osteoporosis and BPPV have been reported (Vibert, et al., 2003). Patients with both osteoporosis and BPPV may be at greater risk for fractures resulting from falls related to BPPV; therefore, patients with combined osteoporosis and subsequent BPPV should be identified and monitored closely for fall and fracture risk. Examined from a different vantage point, patients with a history of recurrent falls, particularly among the elderly, should be assessed for underlying BPPV as one of the potential fall-precipitating diagnoses (Jonsson, et al., 2004).
BPPV may occur in the setting of other CNS disorders. Patients should be questioned as to the presence of preexisting CNS disorders that may modify the management of BPPV. BPPV may occur relatively commonly after trauma or traumatic brain injury (Katsarkas, et al., 1999) (Motin, et al.,

2005). Posttraumatic BPPV is most likely to involve the posterior semicircular canal, and studies indicate that posttraumatic BPPV is significantly more likely to require repeated physical treatments (up to 67% of cases) for resolution compared with nontraumatic forms (14% of cases) (Gordon, et al., 2004). In rare instances, posttraumatic BPPV may be bilateral (Katsarkas, et al., 1999). Because posttraumatic BPPV may be more refractory and/or bilateral, thus requiring specialized treatment, a history of head trauma preceding a clinical diagnosis of BPPV should be elicited (Motin, et al., 2005). Although dizziness in the setting of multiple sclerosis may have a wide variety of etiologies, studies of acute vertigo occurring in multiple sclerosis report that a substantial number of patients may have BPPV with a positive Dix-Hallpike maneuver and successful response to a PRM (Frohman, et al., 2003) (Frohman, et al., 2000). This study suggests that patients with BPPV and an underlying CNS disorder may be successfully diagnosed and treated with conventional methods for BPPV.

Finally, in a small percentage of cases, refractory or persistent BPPV may create difficulties from a psychological and/or social-functional perspective for affected individuals (Gamiz, et al., 2004) (Lopez-Escamez, et al., 2005). Outcomes studies have shown that patients with BPPV exhibit a significant negative quality-of-life impact from the diagnosis compared with the normative population in multiple subscales of the Short Form-36 (Lopez-Escamez, et al., 2005) (Lopez-Escamez, et al., 2003). Patients who have preexisting comorbid conditions may require additional home supervision in the setting of BPPV (Whitney, et al., 2005). This supervision may include counseling about the risk of falling at home or a home safety assessment. In rare cases, patients disabled by BPPV-related vertigo, especially if chronic or refractory, may need home assistance or temporary nursing home placement for their safety.

Conclusie

Niveau 3	BPPD gaat veelal gepaard met comorbiditeit zoals diabetes, osteoporose en vasculaire problematiek. BPPD geeft een verhoogde kans op vallen en dit neemt toe bij comorbiditeit.

Overwegingen

Voordeel: Het behandelplan voor BPPD wordt patiënt-specifiek en patiënten met een verhoogd risico op vallen en valgerelateerde morbiditeit worden geïdentificeerd.

- Nadeel: geen
- Kosten: geen
- Rol van de voorkeur van de patiënt: minimaal

Aanbeveling 6

Artsen dienen factoren die een verhoogd risico op vallen geven uit te vragen; dit beinvloedt de behandelkeus van BPPD (voorkeur voor niet-conservatieve behandeling).

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Hoofdstuk 7 Herevaluatie van de behandeling

Uitgangsvraag 6a:

Is het noodzakelijk om de respons op BPPD behandeling te evalueren?
Patients with BPPV, regardless of initial treatment option rendered, will have variable responses to therapy (Cohen, et al., 2005). The response to therapy may depend on several factors including the accuracy of the diagnosis of BPPV, the duration of symptoms prior to the diagnosis of BPPV, compliance with prescribed therapy, and other factors (Hilton, et al., 2004) (Rupa, et al., 2004). Patients with BPPV should be reassessed within a set time interval after the diagnosis of BPPV for several reasons.

Failure to respond to initial therapy may indicate an initially erroneous diagnosis of BPPV, and one of the major goals of reassessment is to ensure the accuracy of diagnosis of BPPV. As noted, other more serious CNS disorders may mimic BPPV, and these conditions would not be expected to respond to traditional therapies prescribed for BPPV. In cohort studies, the rate of false-positive diagnosis for BPPV subsequently found to be CNS lesions *after failed treatment* (therefore, a highly selected population) with PRM ranges from 1.1 to 3 percent (Rupa, et al., 2004) (Dal, et al., 2000). Thus, persistence of symptoms after initial management requires clinicians to reassess and reevaluate patients for other etiologies of vertigo. Conversely, resolution of BPPV symptoms after initial therapy such as a PRM would corroborate an accurate diagnosis of BPPV.

Patients who are initially treated with vestibular rehabilitation may fail to resolve symptoms owing to multiple factors including poor compliance. In addition, patients who do not respond to initial therapy are likely to remain at risk for falls, decreased quality of life, and other consequences of unresolved BPPV. For these reasons, patients whose symptoms of BPPV fail to resolve should also be identified and classified as initial treatment failures. To define a treatment failure in BPPV, the clinician needs to determine both a failed outcome criterion and an appropriate time interval for assessment of treatment failure. Successful treatment outcomes for interventions for BPPV are traditionally measured in clinical trials by subjective symptom resolution and/or by conversion to a negative Dix-Hallpike test. Almost all treatment trials for BPPV report an outcome measure in the form of the patient's reported symptoms, typically reported among three categorical outcomes: complete resolution of symptoms, improvement, or no improvement/worsening (Hilton, 2004). When included in meta-analyses, treatment responses are typically incorporated as "all or none" for the complete resolution of vertigo (Hilton, et al., 2004) (Woodworth, et al., 2004) (Teixeira, et al., 2006).

Because effective treatment options are available for BPPV that typically render patients symptom free (if treatment is successful), it is logical to use complete symptom resolution as the outcome of choice at the time of reassessment by the clinician. A symptom-based reassessment also allows clinicians to use clinical judgment as to the most appropriate modality for follow-up for individual patients, including telephone communication, electronic communication, or office based reexamination. This symptom-based assessment of treatment resolution should be detailed enough to distinguish patients with truly decreased symptoms related to treatment or patients with minimized symptoms attributable to positional avoidance (who, in fact, may not be treatment successes) from those with true symptom resolution (Woodworth, et al., 2004).

Although conversion to a negative Dix-Hallpike test may have the advantage of being a more objective reassessment than patients' reported symptoms, it also carries the disadvantage of requiring a repeat clinical visit on the part of the patient with associated direct and indirect costs.

The Dix- Hallpike test status is commonly reported in therapeutic trials of BPPV. Persistent symptoms of BPPV and other underlying conditions, however, have been reported in the face of negative Dix-Hallpike testing after therapy, potentially making this a less sensitive reassessment tool (Lynn, et al., 1995) (Magliulo, et al., 2005).

Conversely, patients may report an absence of symptoms after therapeutic intervention yet still have a positive Dix-Hallpike test (Cohen, et al., 2005) (Froehling, et al., 2000) (Sherman, et al., 2001). "Subclinical BPPV" has been offered as an explanation for this (Cohen, et al., 2005). Because of the potential discordance between negative Dix-Hallpike conversion and patients' reported symptoms after treatment for BPPV, Dix- Hallpike conversion is not recommended as the primary reassessment criterion in routine clinical practice but may still be used as a secondary outcome measure. There is no widely accepted time interval at which to assess patients for treatment failure. Therapeutic trials in BPPV variably report follow-up assessments for treatment outcomes at 40 hours, 2 weeks, 1 month, and up to 6 months, although the most commonly chosen interval for follow-up assessment of treatment response is within or at 1 month (Hilton, et al., 2004) (Woodworth, et al., 2004) (Teixeira, et al., 2006). Because the natural history of BPPV exhibits a relatively consistent spontaneous rate of resolution with observation alone, a longer time interval between diagnosis and reassessment would allow patients with true BPPV to resolve symptoms spontaneously, likely irrespective of treatment (Sekine, et al., 2006).

Conversely, the choice of an excessively long time interval between diagnosis and reassessment would also allow cases of an erroneous BPPV diagnosis to potentially progress, leading to potential patient harm. In addition, because recurrence of BPPV may occur as early as 3 months after initial treatment, further delaying the time interval for reassessment may erroneously incorporate a recurrent BPPV syndrome (ie, the initial BPPV responded to treatment with a suitable symptom-free interval thereafter, followed by recurrent BPPV) rather than a persistent BPPV syndrome (Nunez, et al., 2000) (Helminski, et al., 2005).

Given that commonly reported rates of spontaneous complete symptom resolution at the 1-month interval for BPPV range from 20 to 80 percent at 1 month, reassessment at 1 month will also better allow for patients to be reconsidered for further interventional treatment to treat unresolved BPPV (Froehling, et al., 2000) (Lynn, et al., 1995) (Yimtae, et al., 2003) (Munoz, et al., 2007) (Sekine, et al., 2006) (von Brevern, et al., 2006). Thus, choosing a reassessment time interval of 1 month after diagnosis allows a relative balance between overly early reassessment (which would force the unnecessary reassessment of patients who would likely resolve with additional time) and unduly delayed reassessment (which would potentially allow harm from an unknown missed diagnosis or relegate patients to an excess time interval of symptomatic suffering from BPPV). One potential problem with a strict time interval for reassessment is that patients may not have been exposed to their initial treatment (vestibular rehabilitation or PRM as opposed to observation, which may begin immediately after diagnosis) within 1 month of diagnosis depending on referral patterns, patient preferences, or waiting lists for specialty evaluation and treatment. This situation is especially true when the diagnosing clinician may not be the same as the treating clinician. Even if a delay occurs between BPPV diagnosis and completion of the initial treatment, clinicians should still reassess patients at 1 month but may choose to reassign a second time interval for reassessment after completion of the initial treatment option

Niveau 3	Het niet reageren op behandeling kan betekenen dat de oorspronkelijke diagnose niet goed was en dat er sprake is van centrale pathologie. Blijvende klachten betekenen een blijvend risico op vallen en verzuim.

Overwegingen

- Voordeel: identificatie van patiënten met aanhoudende klachten die in eerste instantie met observatie behandeld werden en die zouden kunnen profiteren van de repositiemanoeuvre of een hernieuwde manoeuvre moeten ondergaan. . Identificatie van patiënten waarbij de diagnose herzien moet worden
- Nadeel: geen
- Kosten: kosten van herbeoordeling
- Afweging: het voordeel weegt op tegen het nadeel.
- Waarde oordeel: bevestiging van de diagnose en het ondervangen van patiënten die zouden kunnen profiteren van een andere behandeling.
- Rol van de voorkeur van de patiënt: minimaal

Aanbeveling 6

Een maand na de behandeling dient het effect van de behandeling geëvalueerd te worden.

Uitgangsvraag 6 B:

Hoe zou de evaluatie van de BPPD behandeling eruit moeten zien?

Although the repositioning maneuvers have a substantial success rate, they are not expected to solve the at yet unknown origin of clod or debris formation, which may explain the substantial number of re/occurrences of BPPV. Patients with persistent symptoms of vertigo, dizziness, or unsteadiness at the time of reassessment of the initial treatment response are classified as treatment failures. Treatment failures require reevaluation for the following reasons: 1) Persistent BPPV may be present and responsive to additional maneuvers; 2) coexisting vestibular conditions may be present that can be identified and treated; and 3) serious CNS disorders may simulate BPPV and need to be identified (Furman, et al., 1995) (Furman, et al., 1999) (Rupa, et al., 2004).

<u>Persistent BPPV</u>

Patients with BPPV who initially are treated with observation may fail to resolve spontaneously and have persistent BPPV at the time reassessment. Also, on the basis of failure rates of vestibular rehabilitation or a single-session PRM ranging from 15 to 50 percent, a significant number of patients initially managed with vestibular rehabilitation or PRM will have persistent BPPV at reassessment, which also indicates a treatment failure Furman, et al., 1999) (Hilton, et al., 2004) (Cohen, et al., 2005) (Teixeira, et al., 2006) (von Brevern, et al., 2006). Reevaluation of a treatment failure should include obtaining a history of vertigo, determining if the vertigo is provoked by positional change relative to gravity (ie, lying down in bed, rolling over, bending down, or tilting the head back), which then suggests persistent BPPV. As with the original diagnostic criteria, the Dix-Hallpike test should be repeated to confirm the diagnosis of BPPV. If the Dix-Hallpike maneuver is still positive, repeat PRMs

can then be performed as a preferred treatment. The rate of successful treatment of BPPV reaches 90 to 98 percent when additional repositioning maneuvers are subsequently performed (Brocchetti, et al., 2003) (Beynon, et al., 2000). Therefore, the PRMs are the treatment of choice for initial BPPV treatment failures deemed to be due to persistent BPPV.

A similar approach may be adopted for the reevaluation of persistent symptoms of vertigo after an initial diagnosis of lateral canal BPPV. The supine roll test should be repeated and, if characteristic nystagmus is elicited, a PRM appropriate for lateral canal BPPV may be repeated as well. There are limited data regarding the management of treatment failures after PRM for lateral canal BPPV, because this condition seems to respond more consistently to PRM and it also has a higher spontaneous resolution rate (Tirelli, et al., 2004) (Sekine, et al., 2006) (Fife, et al., 1998) (Asprella Libonati, et al., 2005). Some studies indicate cure rates of 86 to 100 percent with up to four PRM treatments in lateral canal BPPV (Casani, et al., 2002) (Chiou, et al., 2005). Further subanalysis suggests that the apogeoptropic variant of lateral canal BPPV may be more refractory to therapy (White, et al., 2005) (Casani, et al., 2002).

A small percentage of patients initially diagnosed and treated for lateral canal BPPV or horizontal canal BPPV may experience a canal switch. In these cases, initial horizontal canal BPPV may transform into posterior canal
BPPV in up to 6 percent of cases (Nuti, et al., 1998) (Tirelli, et al., 2004). Similarly, a small fraction of patients (also approximating 6%) initially presenting with posterior canal BPPV may transition after treatment to lateral canal BPPV (Yimtae, et al., 2003) (Herdman, et al., 1996). A small subset of patients who do not respond to treatment for posterior canal and/or lateral canal BPPV may suffer from anterior canal BPPV, and may need to be evaluated accordingly (Jackson, et al., 2007). Finally, although rare, two semicircular canals may be simultaneously involved. The second canal's involvement may become evident at the time of reassessment if one of the involved canals was appropriately treated (Rupa, et al., 2004). Thus, reassessment of persistent positional vertigo in BPPV should include examination for involvement of other semicircular canals than originally diagnosed. Coexisting Vestibular System Dysfunction A BPPV treatment failure subsequently may be found to be a case manifesting vertiginous symptoms that are provoked by head and body movements in general (ie, not primarily provoked by positional changes relative to gravity); unprovoked (ie, spontaneous) episodes of vertigo occurring while not moving; or in fact, a constant unsteadiness. These specific findings should be identified by clinicians at the time of reevaluation; such findings suggest the presence of vestibular system dysfunction associated with or in addition to the initially treated BPPV. There may be several possible factors at play when vestibular system dysfunction accompanies BPPV.

In a study by Monobe et al, (Monobe, et al., 2001) treatment failure of the PRM was most commonly seen in patients with BPPV secondary to head trauma or vestibular neuritis. Because vestibular neuritis and head trauma are both frequently associated with vestibular dysfunction, the cause of persistent symptoms following treatment of BPPV is likely related to widespread dysfunction within the vestibular system in this setting (Bergenius, et al., 1999). Because BPPV is more common in patients with Ménière's disease and migraine, vestibular system dysfunction associated with these disorders can lead to prolonged symptoms of BPPV, greater chance for recurrence of BPPV, and increased risk for falls, particularly in older persons (Gordon, et al., 2004) (Roberts, et al., 2005) (Hughes, et al., 1997) (Dornhoffer, et al., 2000) (Uneri, et al., 2004) (Kayan, et al., 1984).

In addition, BPPV not associated with any other ontological or neurological disease can still be associated with an underlying impaired vestibular function, and these individuals are more likely to have incomplete resolution of symptoms even if their Dix-Hallpike testing normalizes with PRM (Pollak, et al., 2002). Finally, transient vestibular dysfunction can also occur following repositioning maneuvers. Evidence suggests that balance function continues to be affected between 1 to 3 months after repositioning maneuvers, and that some of these patients may need additional balance therapy (ie, counseling, vestibular rehabilitation) to prevent falls and decrease their fear of falling after the vertigo from BPPV has resolved (Blatt, et al., 2000) (Chang, et al., 2006) (Giacomini, et al., 2002) (Black, et al., 1984). Thus, reevaluation of BPPV treatment failures should include a search for these associated conditions.

When coexisting vestibular system dysfunction is suspected, additional testing should be considered. This testing may include audiometric testing to screen for Ménière's disease and nerve VIII pathology such as acoustic neuroma, vestibular function testing to detect central and peripheral vestibular dysfunction, and CNS imaging to detect CNS pathology. Such subsequent testing will need to be tailored to the clinical presentation, and clinicians should exercise their clinical judgment. Vestibular rehabilitation has been shown to be an effective treatment for vestibular symptoms due to the potentially persistent vestibular dysfunction associated with BPPV; this treatment may reduce the risk for falls (Angeli, et al., 2003).136

CNS Disorders Masquerading as BPPV Although vertigo of central origin is frequently associated with neurological symptoms such as gait, speech, and autonomic dysfunction, it is important to recognize that, rarely, CNS disorders can masquerade as BPPV (Bertholon, et al., 2002). Many of these have been previously discussed in the section on differential diagnosis, but the relative likelihood of their diagnosis increases in the face of initial treatment failure. In one study, a CNS disorder that explained BPPV treatment failure was found in 3 percent of patients (Dal, et al., 2000).

Whenever the signs and symptoms of BPPV are atypical or refractory to treatment, additional history and physical examination should be obtained to address the possibility of undiagnosed CNS disease (Smouha, et al., 1995). Patients with symptoms consistent with those of BPPV who do not show improvement or resolution after undergoing the PRM, especially after two or three attempted maneuvers, or those who describe associated auditory or neurological symptoms should be evaluated with a thorough neurological examination, additional CNS testing, and/or MRI of the brain and posterior fossa to identify possible intracranial pathological conditions (Dunniway, et al., 1998) (Buttner, et al., 1999).

Conclusie

Niveau 4	Bij falen van de behandeling kan er sprake zijn van 1) persisterende of andersoortige BPPD 2) co-existente vestibulaire aandoening 3) centrale aandoening

Overwegingen

- Voordeel: het opstellen van een effectief behandelplan voor patiënten met persisterende BPPD en comorbiditeiten, afname in de kans op het missen van ernstige aandoeningen die een andere behandeling vereisen.
- Nadeel: geen
- Kosten: kosten van herevaluatie en vervolgtesten
- Afweging: de voordelen wegen op tegen de nadelen.
- Waarde oordeel: behandeling van BPPD en andere vestibulaire aandoeningen die in combinatie kunnen voorkomen; behandeling van persisterende BPPD met een repositie manoeuvre na expectatief beleid of vestibulaire revalidatie.
- Rol van de voorkeur van de patiënt: minimaal

Aanbeveling

Bij falen van de behandeling moet geëvalueerd worden of de patiënt 1) persisterende of andersoortige BPPD heeft 2) co-existente vestibulaire aandoeningen heeft die geïdentificeerd en behandeld moeten worden en 3) een centrale aandoening heeft.

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Hoofdstuk 8 Voorlichting patiënten

Uitgangsvraag 7:

Ten aanzien van welke aspecten rond BPPD zouden patiënten voorgelicht moeten worden?

Although BPPV generally responds well to treatment, there is a significant rate of BPPV recurrence after initial resolution or clinical cure. Most trials of BPPV maintain limited follow-up, rarely beyond 3 months. In the few trials of BPPV with longer-term follow-up, the rate of recurrent BPPV (ie, BPPV symptoms manifesting again after a symptom- free period) is reported to be 5 to 13.5 percent at 6-month follow-up (Macias, et al., 2000) (Sridhar, et al., 2005). At 1 year after treatment, the rate of recurrence has been reported at a slightly higher rate of 10 to 18 percent (Prokopakis, et al., 2005) (Sakaida, et al., 2003). The recurrence rate continues to increase and may be as high as 37 to 50 percent at 5 years by Kaplan-Meier estimation (Nunez, et al., 2000) (Sakaida, et al., 2003). Overall the recurrence rate of BPPV may be estimated at 15 percent per year (Nunez, et al., 2000). Patients with BPPV after trauma are likely to demonstrate an even higher recurrence rate of their BPPV (Gordon, et al., 2004). Thus, clinicians should be aware of the recurrence risk of BPPV and should counsel patients accordingly. Counseling will likely have several benefits, which include earlier recognition by patients of recurrent BPPV, allowing earlier return for PRM or vestibular rehabilitation. Also, counseling regarding recurrence will offset the potential anxiety patients may feel when BPPV recurs and allow them to make corresponding adjustments in their daily routine to minimize the impact of BPPV symptomatology. As with any balance or vestibular disorder, patients with BPPV should be counseled regarding the potential that BPPV may place them at greater risk for falls (Brandt, et al., 1993). This risk may apply particularly to patients with preexisting balance disorders or vestibular deficits and a separate onset of BPPV. The propensity for falling may actually be a significant motivating factor for patients to be referred for evaluation of underlying BPPV (Lawson, et al., 2005). The risk of falls and fear of falls are significant considerations in the management of the elderly who suffer from chronic dizziness (Gazzola, et al., 2006). In a study of 120 elderly patients with chronic vestibular disorders, 36.7 percent carried the diagnosis of BPPV. Fifty-three percent of subjects had fallen at least once in the past year, and 29.2 percent had recurrent falls (Gazzola, et al., 2006). Other authors have confirmed a relatively high rate of BPPV and associated falling tendencies in the elderly (Oghalai, et al., 2000) (Imbaud Genieys, et al., 2007).

Practically speaking, clinicians should counsel patients and their families regarding the risk of falls associated with BPPV. This information is particularly important for the elderly and frail who may be more susceptible to serious injury as a result of falling. Such counseling could include assessment of home safety, activity restrictions, and the need for home supervision until BPPV is resolved (Rubenstein, et al., 2006). Patients may be particularly vulnerable in the time interval between initial diagnosis of BPPV and definitive treatment when they are referred to another clinician for PRM or vestibular rehabilitation. Counseling should therefore occur at the time of initial diagnosis. The costs of such counseling are anticipated to be minimal and will enhance patient and public safety while avoiding potential posttraumatic sequelae.

Finally, patients should be counseled regarding the importance of follow-up after diagnosis of BPPV. Patients initially treated with observation should be counseled that, if BPPV fails to resolve spontaneously, effective therapies such as the PRM may then be undertaken. Also, patients should be educated about atypical symptoms (subjective hearing loss, tinnitus, aural fullness, gait disturbance, non-positional vertigo, nausea, vomiting, etc.) whose occurrence or persistence after resolution of the primary symptoms of BPPV warrant further clinical evaluation (Rupa, et al., 2004). As noted, such symptoms, particularly when unmasked by the resolution of BPPV may indicate an underlying vestibular or CNS disorder. Clinicians may also educate patients with refractory BPPV or repeatedly recurrent BPPV that in select cases a surgical remedy ("canal plugging procedure" or singular neurectomy) may be considered (Parnes, et al., 2003) (Shaia, et al., 2006).

Niveau 2/3?	Er is bij BPPD sprake van een verhoogde valkans alsook angst om te vallen. Er is tevens een aanzienlijke kans op recidief BPPD na behandeling.

Overwegingen

- Voordelen: toename van bewustwording van valrisico, afname van verwondingen door vallen, toename van bewustwording bij patiënten van het risico op terugkeer van BPPD, waardoor ze snel opnieuw voor behandeling komen.
- Nadelen: geen
- Kosten: geen
- Afweging: De voordelen wegen op tegen de nadelen.
- Rol van de voorkeur van de patiënt: geen

Aanbeveling 7

De patiënt dient verteld te worden dat BPPD een goedaardige, meestal goed behandelbare aandoening is. Het is belangrijk dat de patiënt (eventueel ook de familie) goed voorgelicht wordt over de risico's op vallen bij BPPD, de aanzienlijke kans op een recidief BPPD en het belang van controle door de behandelaar.

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