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MULTI-MODAL APPROACH TO SEVERE REFRACTORY MOTION SICKNESS IN A FAMILY OF COMMERCIAL AIRLINE PASSENGERS

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ABSTRACT BACKGROUND

Motion sickness affects at least one third of the population, varying in degree of individual sensitivity and severity. Sensory mismatch between vestibular, visual and proprioceptive inputs with the cognitive visuospatial map is the primary theory for its occurrence. Whilst stimulus avoidance and medication are common treatment, neurovestibular habituation is often overlooked as therapy in civilian patients.

A 55-year-old woman presented with her two adult children with a life-long history of severe motion sickness. Significant anticipatory anxiety accompanied with nausea would begin prior to boarding, triggered by the smell of airline food and jet fuel. In flight, dizziness and severe recurrent vomiting would occur, often leading to incapacitation and pre-syncope. Specialist investigation found no underlying vestibular or neurological disorder. A multi-agent pharmaceutical approach with neurovestibular habituation exercises allowed the patient to undertake a multi-stage international airline travel without significant illness. Her daughter reported similar improvement on medication alone. Following a break in habituation exercises and without the medication, the symptoms reoccurred on a future flight.

Motion sickness is often treated as a vestibular condition in isolation without consideration of the neuropsychological aspects. Conditioning to environmental cues including smells along anticipatory anxiety may trigger symptoms of motion sickness in absence of motion cues. Multi-agent pharmaceutical therapy may be more effective than single agents alone**.** Neurovestibular habituation exercises are low cost but effective means of producing tolerance of motion cues with symptom reduction. This can be sustained with ongoing environmental exposure.

KEY WORDS

Motion sickness; vestibular; habituation exercises; anti-emetics

BACKGROUND

Motion sickness is common condition, occurring in up to 30-46% of the population[1, 2], with a considerable degree of individual variation in sensitivity and severity. The interpersonal variation is often explained due to difference in neuronal development, otolith asymmetry, genetic polymorphisms[1] or psychological conditioning[3, 4]. However, the theory of sensory conflict, which postulates motion sickness occurs due to a mismatch between cognitive visuospatial mapping and vestibular, visual and proprioceptive inputs, is the most accepted explanation[2, 5, 6]. Motion sickness can also be triggered in the absence of a vestibular input, which is become more prevalent with increasing use of virtual reality technology[2]. Yet, motion sickness is often considered and treated as a vestibular problem in isolation.

This family case study follows a 55-year-old female and her two adult children who presented to an aviation medicine clinic, requesting assistance prior to a long-haul flight for a life-long history of severe refractory motion sickness. The symptoms often developed prior to boarding and would intensify during flight leading to incapacitation. Despite extensive investigation, no underlying physical pathology was identified. Progressive neurovestibular desensitization training, along with a multi-agent pharmacological approach, resulted in complete resolution of symptoms. This case highlights that once organic pathology is excluded, neurovestibular and psychological therapy should be considered alongside a multi-agent pharmacological approach in managing severe motion sickness.

CLINICAL RECORD

A 55-year-old female self-referred to an aviation medicine clinic in Australia with a life-long history of severe motion sickness. She first encountered it as a child during road and rail trips through the mountains of Europe. She had never been able to tolerate any form of sea travel and whilst she could tolerate air travel as a child, she now experienced severe and disabling symptoms. As a result, she had avoided travelling however her family wished to travel back to visit the extended family in western Europe.

Often the symptoms occurred pre-flight with anticipatory nausea, sweating and anxiety. Olfactory cues such as airline food and aviation fuel were common triggers. Once a flight commenced, the symptoms intensify and rapidly lead to vomiting. Twice she experienced pre-syncopal episodes requiring medical assistance on arrival at the layover destination. No dizziness, vertigo or tinnitus was experienced. Of note, her adult daughter and son also experienced moderate to severe motion sickness, with similar pre-flight anticipatory anxiety and nausea. However, they both stated that their motion sickness was often amplified once their mother becomes unwell.

Previously she had trialled common countermeasures as single agents including hyoscine tablets and patches, ginger, promethazine and compression bands to no effect. After extensive investigation by an ENT specialist with audiometry, tympanometry and MRI scan, no underlying vestibulocochlear abnormalities were identified. She had no relevant past medical history and did not take regular medication.

On examination, her blood pressure was 134/87mmHg, heart rate 70 beat per minute and BMI of 20. ENT exam was unremarkable with no evidence of middle ear disease. Ophthalmic and neurological assessment were unmarkable with no tremors, limb weakness, ataxia, ophthalmoplegia or field defects. Head impulse, nystagmus, test of skew examination and Romberg tests were negative. Dix-Hallpike test was normal, but provoked nausea.

She was referred to a specialist physiotherapy dizziness clinic for neurovestibular habituation training. These exercises were modified from Rine et al[7], involving the patient fixating at arm's length on their index finger, which is then moved along the horizontal plain at increasing velocity until symptoms occur. The exercises are then repeated in the arm moving in vertical axis, followed by keeping the arm stationary with the head moving in horizontal, vertical and rotational axes respectively. As the first session provoked severe nausea, she was prescribed prochlorperazine 5mg three times a day to start the day prior to a session. This allowed her to tolerate the physiotherapy sessions and thus was able to continue these exercises daily at home.

One day prior to flying, she was advised to commence prochlorperazine. Lorazepam 1mg was to be taken 1 hour prior to flying to reduce anticipatory nausea and anxiety. Ondansetron 8mg PRN was provided as an adjunct in the event of nausea developing in flight. Both her children were prescribed the same regime but opted not to undertake habituation training.

Following two months of habituation training at home and by using the medication regime, the patient was able to undertake a four leg return flight to Europe. She reported a complete resolution of symptoms and was able to enjoy flying for the first time without vomiting. Her daughter, who took the same medication, noted no symptoms prior to or during the flight. Her son was reportedly highly anxious pre-flight and thus still experienced nausea and vomiting in flight. On follow-up eighteen months later, she reported that since ceasing the exercises and not taking the medication for the flight, the symptoms reoccurred with the same severity. This included another syncopal episode, which following further investigations again revealed no cardiovascular, ENT or neurological cause for her symptoms.

DISCUSSION

This family case study demonstrates the utility of a multimodal approach to severe motion sickness. Whilst there is suggestion of familial pre-disposition to motion sickness, this family had a strong conditioned response characterised by significant anticipatory anxiety existed that amplified the symptoms. By undertaking habituation training, with the assistance

of targeted multi-agent pharmacotherapy, a complete resolution of symptoms was achieved during the initial period of therapy for the mother. With the removal of triggering cues of her mother being unwell and a reduction in anticipatory anxiety, the daughter also achieved symptom control.

Traditional practice suggests the avoidance of the provocative motion cues[6], combined with anticholinergic and antihistamine medication that inhibit the vestibular system, such as hyoscine and promethazine, as the most effective treatments[2, 5, 8]. Whilst effective at reducing symptoms, hyoscine also shortens the habituation time required in ab initio aviators[9]. Historically, prochlorperazine has also been trialled but considered ineffective as a sole agent[8]. Dopaminergic and serotonergic anti-emetics including metoclopramide and ondansetron are ineffective at vestibular suppression but may reduce symptoms and prevent vomiting [5].

The psychological component is often overlooked in motion sickness, particularly in severe cases that are refractory to medication. Repeated exposure to environmental cues, such as petrochemical fumes or food, during an episode of motion sickness may condition a future symptomatic response even in the absence of vestibular input. Memories of previously traumatic episodes of motion sickness may also trigger anticipatory anxiety and amplify somatic symptoms[3], particular in individual with high trait anxiety[4]. Whilst the use of a short-acting anxiolytic was effective in this case, some patients may require psychotherapy to address the conditioning.

Habituation training is commonly used for military aircrew who suffer from motion sickness, often identified during ab initio training[10-12]. Multi-axis stimulation of the semicircular canals, with increasing amplitude and frequency across numerous sessions, has been shown to produce a significant improvement in tolerance of motion and reduction in symptoms[10]. Medication is often used in facilitating this habituation[9]. This can be achieved using a protocol of increasing intensity on a motorized Bárány chair[10, 12] or patented exercise programs such as the Puma method[11].

Neurovestibular habituation exercises involving head tracking and target fixation can be an effective, low-cost option can be conducted at home without specialist equipment[2, 7]. These exercise attempt to induce temporary threshold shift for the onset of symptoms by creating sensory conflict and forcing an adaption of the cognitive visuospatial map. Patients focus on a target, with either the object or the patients head moving at a steady reversing velocity along the horizontal axis. Through repeated sessions and across multiple axes, the movement rate is increased gradually until the threshold of symptoms is encountered. Patients are then instructed to continue at this threshold until symptoms are no longer tolerable before ceasing for a break. Sessions are conducted as often as tolerated with the aim to eventually increase the threshold at which symptoms occur. The Puma method is based on the same underpinning theory however it combines this with full body movements and involves cross-coupling of the semi-circular canals[11], which may create a more provocative response.

It is important to note that habituation training only results in a temporary threshold shift. Once satisfactory control is achieved, pending the patient continuing to be exposed to proactive environmental cues, the habituation appears to persist. However, upon ceasing regular exposure, either through removal from the stimulating environment or by discontinuing habituation training, patients will regress towards their pre-invention symptoms. The author notes anecdotally that such an experience is familiar amongst aerobatic and military aviators who notice a decrease in their tolerance following an extended break from flying. Despite the success of the initial therapy period in the mother, since the exercises were not sustained or reinitiated for later flights, her motion sickness reoccurred with the same severity.

This case study demonstrates that a multi-modal approach to motion sickness, particularly in severe cases, can result in a satisfactory outcome for patients. When avoidance of provocative motion cues is not possible and single agent pharmacotherapy is ineffective, the psychological and neurovestibular components must be addressed. Whilst in contemporary practice neurovestibular habituation is often overlooked, it offers a low-cost, simple and potentially effective therapy option for patients suffering from motion sickness. This is particularly true when combined with multi-modal pharmacotherapy to facilitate the habituation process as well as addressing anticipatory symptoms and anxiety.

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