Motorist's vestibular disorientation syndrome

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SUMMARY Six patients are described who experienced difficulty in driving a motor car. Four had illusions that the car was turning, which occurred particularly on open, featureless roads or the brows of hills and caused the driver to stop. All patients had peripheral or central neuro-otological abnormalities, but the only finding consistent with the directionality of the symptoms was an unpleasantly increased sense of circularvection during optokinetic stimulation in the direction of the illusion. These problems occur because of a false sense of orientation arising either from inappropriate signals from disordered vestibular canal and otolith organs or from a disordered central interpretation of vestibular information, and become manifest in the absence of adequate visual stabilisation. The other two patients with lateralised vestibular disease made inappropriate steering adjustments in the direction of the imbalance of vestibular tone.

False perception of the motions of the self and/or external objects in the physical world may be provoked by neurological disease or by unusual conditions of motion and environmental structure. For example, peripheral vestibular disease may result in the illusion that self and the environmental surrounds are rotating. In contrast, a normal subject whilst flying an aircraft, blind in cloud, may feel that he is flying on a straight course when in fact he is in a prolonged turn.¹ In both cases, the sensory inputs which are normally combined to provide an accurate model of the physical world provide false information because either the stimuli are unphysiological or important sensory information is lacking or distorted. The resulting misleading sense of movement and disorientation may be associated with other somatic symptoms such as nausea, sweating and a sense of anxiety and panic.² This article documents an unusual group of four patients with minimal symptoms of neuro-otological disease who become disorientated when they drive a car in particular environmental conditions. For comparison, two others in whom steering was impaired by vestibular disease are described. The four patients presented to

the Neuro-otology clinic of the National Hospital with the specific complaint of difficulty in steering a car. The neuro-otological abnormalities are summarised in the table.

Case reports

Case 1

A 43-year-old female presented with difficulty driving for 4 years. She had first noticed that the car seemed to pull toward the left on open roads and in the absence of other vehicles. During this she had a sensation of falling to the right, leaned to the left in an attempt to compensate and then developed palpitations, nausea, panic and disorientation. Mechanical error was excluded and similar episodes became more frequent and occurred at lower speeds and on larger roads in towns. The symptoms were readily provoked on right hand bends, especially on roads with marked camber. The symptoms would also occur going over the brow of or down hills. Eventually, she could only drive comfortably at low speeds and her daily activities were restricted. Neurological, and psychological (Prof I Marks) assessments were normal. Neuro-otological testing showed a negative Romberg Test and normal gait but she turned to left on the 2-step test. There was a 2° nystagmus to the right with removal of fixation. Horizontal pursuit was bidirectionally hypometric with saccadic corrections. VOR responses to sinusoidal rotatory stimuli were normal with a slight preponderance of right beating response. Vestibular ocular reflex suppression (VORS) was impaired

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Table Neuro-otological findings

Age (yr) Sex	Type of disorientation	Associated symptoms	Predisposing factors	Duration
Case 1 43 F	Car pulling to L.	Tilt to R Nausea, Panic, Disorientation	R. hand bends Open bends.	4 yr
Case 2 43 M	Car turning to R	Tilt to R Nausea, derealisation	L. hand bends, Motorways, Downhill.	4 yr
Case 3 62 F	Car pulling to R	Tilt to R "Insecurity" Disorientation	R. hand bends Open roads, Brow of hill.	6 yr 4 yr BPV
Case 4 48 M	Car deviating to R	"Force" to R, Gap to door, panic Veers to L walking, Visual vertigo	Speed Open roads	15 yr
Case 5 58 M	Drives car to L		Continuous	4 yr
Case 6 50 M	Drives to L	Vertigo	Continuous	2 yr

DP = Directional preponderance

R = Right

L = Left

->L = Sways or veers to left

CP = Canal paresis

BPV = Benign positional vertigo

N = Negative

bidirectionally with no effect of alteration of background characteristics. Optokinetic nystagmus and its suppression by fixation were normal; however, stimulus movement to the right appeared faster, and the illusion of self-rotation or circularvection was stronger, causing nausea and disorientation.

Case 2

In 1980, a 43-year-old man developed unpleasant symptoms while driving. When turning bends to the left the car would seem to continue straight ahead. He had the car checked and even changed cars but the episodes became more frequent, especially on wider roads, particularly motorways, at higher speeds and on curves to the left. He felt loss of control, a sensation of his body falling to the right and would develop sweating, panic and a sense of derealisation. The attacks have persisted to the present time with variable degrees of provocation. They always occur above 70 mph (112 km/h) but also at about 40 mph (64 km/h) and are worse at night. Surroundings such as buildings, trees and hedges give him a feeling of security and he is always worse on motorways and may have to stop; he can only avoid stopping when he knows that a right hand bend (which will counteract the symptoms) is approaching. Descending after the crest of a hill is a further exacerbating factor. He has had similar, but milder sensations as a passenger and the symptoms have not occurred whilst travelling on a train. He had never suffered vertigo, tinnitus or deafness. Treatment with cinnarizine had not relieved his

symptoms. General examination was normal, Romberg negative and gait with eye closure was unsteady. Investigations in 1980 had suggested a central vestibulo-cerebellar disturbance, particularly involving the left side (Dr MR Dix). Recent testing revealed second degree nystagmus to the left. Pursuit to right and left was mildly impaired with saccadic intrusions. Responses to full field optokinetic stimulation were normal, however circularvection elicited by the leftward stimulus was faster and provoked dizziness, nausea and disorientation. Suppression of optokinetic nystagmus by a fixation point and optokinetic after nystagmus were normal. VOR responses to sinusoidal rotation in the horizontal plane were 50% of normal duration but symmetrical. VORS was bidirectionally impaired.

Case 3

A 62-year-old female first presented in 1976 with a 4 year history of benign positional vertigo in which attacks of rotational vertigo lasting up to five seconds occurred most nights on turning over to the right in bed and were rapidly reversed by turning to the left. These symptoms ceased spontaneously in 1979. Her difficulty with driving first occurred on a dual carriageway that curved to the right; she had to stop the car because it seemed to be veering excessively to the right and she thought she was going too fast. Several months later she had a second, identical attack on the same stretch of road. After some months, on a dual carriageway that had just become three lanes, she had a sudden feeling of insecurity

Signs					Presumed site of lesion
Romberg/Gait	Spontaneous nystagmus	Induced nystagmus	Pursuit nystagmus	Optokinetic nystagmus	
N	2nd° - R	DP - R. (Rotation)	Impaired R & L	Drum R Stronger	L vestibular, prob. central.
N	2nd° - L	N	Impaired R & L	Drum L Stronger	R vestibular, prob. central.
N	2nd° - R	DP - R. (Caloric) (Rotation)	N	Drum L Stronger	L vestibular
N	1st° - L	DP - L (Caloric)	—	—	R vestibular
->L	N	CP - L DP R (Caloric)	—	—	L peripheral
->L	Torsional Nystagmus	DP - R (Caloric) (Rotation)	Impaired R & L	DP - R	L central - infarct

and disorientation and the car felt as though it was pulling to the right. Her body felt tilted to the right although she was leaning to the left. One week later she felt as though she was losing control of the car as she drove down a straight hill. On a hilly stretch of roads she had to keep stopping as she drove over the brows of hills. These symptoms, which began in 1976 have persisted with fluctuation ever since. They are more likely to occur on open roads than in towns or traffic. The presence of buildings and high hedges is helpful; she is worse at speed and on right hand bends and now drives at a maximum of 40 mph (64 km/h). The symptoms have occurred when she is a passenger, and then she is worse if she closes her eyes. The symptoms can only be relieved by slowing down. When first seen in 1976, the caloric responses showed a left canal paresis combined with a directional preponderance to the right and electro-nystagmography only revealed nystagmus to the right in darkness. At recent review, Romberg was positive and she veered to the left on gait and two step testing in the dark. There was now second degree nystagmus to the right in the dark and pursuit was normal. Optokinetic responses to full field stimulation were symmetrical, however circularvection was much more unpleasant when the drum turned to the left than the right. Optokinetic after-nystagmus was increased after drum rotation to the left than the right. Optokinetic nystagmus could be suppressed normally by a point fixation. There was a directional preponderance of VOR responses to rotational testing to the right and VORS was normal and not influenced by background variation.

Case 4

A 48-year-old man first attended in 1976. He stated that for 7 years he had suffered from episodes that, when driving, he felt that he was deviating to the right; since that time he had been unable to drive above 45 mph (72 km/h). Above this speed he feels a force pushing the car to the right and has to stop. In addition he experiences a sensation that there is a gap of one or two feet between himself and the door, but with no sensation of body tilt. At times he was only able to drive at 10 mph and the symptoms are equally bad when he is a passenger. These difficulties are worse when driving in darkness or in open country and better in built up areas. In 1976 neuro-otological examination showed normal pure tone audiometry. He had first degree nystagmus to the left and a directional preponderance to the left of optokinetic nystagmus. In darkness the caloric responses showed a pronounced directional preponderance to the left. CT scan of the brain was normal. When reviewed in 1980 these findings were unchanged.

Case 5

A 58-year-old man had experienced intermittent attacks of acute vertigo over the preceding ten years. For three months he had very frequent episodes of mild vertigo precipitated by head movement and felt continuously unsteady, tending to veer to the left. During this time he had considerable difficulty driving as the car tended to pull to the left such that he often bumped the kerb. Bright oncoming headlights and road lights caused nausea, giddiness and dis-

orientation. He had to stop the car and has now given up driving. He was mildly hypertensive but otherwise general and neurological examinations were normal. On neuro-otological assessment, he went to the left on Romberg and gait testing and calorics suggested a left peripheral lesion.

Case 6

In 1982, a 50-year-old man developed a severe headache with acute rotational vertigo. He fell to the left and had marked oscillopsia, particularly on left gaze. He improved over a 6 week period but has remained incapacitated by poor balance; he has a sensation of being pushed to the left and occasionally falls to that side. Whenever he has tried to drive he feels a force pushing him to the left and repeatedly bumps the nearside kerb. These sensations never occur when he is a passenger but he then suffers from distress brought about by a sense of inappropriate, vivid or blurred background movement and nausea. At neuro-otological examination he fell to the left on Romberg and gait testing. There was a clockwise torsional nystagmus in all positions of gaze and a directional preponderance to the right of responses to optokinetic, rotational and caloric testing. Neurological investigations in 1984 were normal and he was presumed to have had a low brain stem infarct.

Discussion

Six patients with neuro-otological disease are described in whom difficulties in driving a motor car constituted the major part of their symptomatology. This was the only complaint in three patients and one had other vestibular symptoms in the past only. The patients can be divided into two distinct groups; in cases 1–4 there was an overwhelming *illusion* that the vehicle was going off course, causing strong somatic symptoms of disorientation, nausea and panic. In contrast, in cases 5 and 6 the vehicle physically moved across the road in the direction appropriate to the patients vestibular disturbance (that is in the same direction as the Romberg and gait) without associated somatic symptoms.

In the first four patients, the driving disturbance had consistent characteristics; the illusion of veering to the side was provoked preferentially when driving on open roads such as motorways with flat and distant borders. Conversely the illusion was suppressed when driving in built-up areas, in traffic or on roads with close high edges (hedges). Three patients experienced symptoms going over the brows of hills and descending hills; one could control the symptoms only if she knew the road and that the downward hill was not too long, as could another patient

if he knew that a favourable corner was approaching. All these patients were worse at higher speeds although the maximum tolerable speeds on any particular type of road could vary from week to week. Patients were able to drive and corner faster in built-up areas than in open areas. Patient 1 could drive at 40 mph in built-up areas but only 25 mph on bends on open roads; patient 4 could, at times, only drive at 10 mph. In each patient, the side to which the car seemed to veer and the direction of corners precipitating the symptoms remained constant. All patients thought the car was physically turning but only in patients 5 and 6 were the cars known to have moved off course. It was initially difficult to be certain about this point because, at first, all the patients were adamant that there was real unwanted movement of the car (two patients had their cars checked or changed) until they realised that the symptoms were being caused by an illusion. Three patients experienced a strong sensation of body tilt which they eventually realised initiated an illusion of turning. Eye closure (when a passenger) did not relieve the symptoms and three patients were worse with eye closure or in the dark.

Neuro-otological testing revealed abnormalities in each case; unidirectional spontaneous nystagmus in five, abnormalities of calorics in four and impairment of smooth pursuit in two. Pursuit abnormalities indicate a central lesion, however, in cases 3 and 4 it is difficult to localise the lesion with certainty. In the three patients tested by full-field optokinetic stimuli, the induced nystagmus was normal and symmetrical, however the subjective sensation of drum velocity and self-rotation was greatly increased and unpleasant when the circularvection was in the same direction as the perceived movement of the car off course. This was the only finding that corresponded with the directionality of the illusion.

Disorientation whilst driving is not a common complaint in patients with neuro-otological disease and this symptom is evidence of a particular set of abnormalities which render the patient susceptible. The common factors involved in the genesis of this disorientation syndrome are the proximity and quality of the background surroundings and the speed and direction of the vehicle. Near, highly structured background or the presence of other traffic has a protective effect on the individual's ability to corner or drive fast.

A theoretical explanation can be proposed to account for the illusions of turning, tilt or the feeling of being pushed sideways. Of first consideration, driving a car involves unusual visual and motion stimuli which do not occur in normal activities. Much of the vestibular stimulation involved in driv-

Fig Forces acting and sensations perceived during cornering (after Guedry⁴). The convention for indicating otolith signals is a vector in the direction in which the head feels it is accelerating. The convention for indicating the direction of the gravity vector (G) is in the form of an acceleration in the upwards direction. (The force of gravity (FG) pulling the body downwards is the same as one would experience standing on a platform away from any gravitational field which was accelerating at $1G$ upwards). (A) Normal driver seen from behind when turning to his right, without tilt of the vehicle or driver. The linear forces experienced are G and a centrifugal force (CF) which arises from the centripetal acceleration (CA). The otoliths would signal an acceleration vector (OT). If a canal signal indicating tilt is absent the PREDOMINATE subjective interpretation is of centripetal acceleration/centrifugal force and not of a tilted acceleration vector. (B) A more realistic interpretation of cornering in which the driver turns to the right, the vehicle tilts to the left under centrifugal force and the driver leans into the corner. Both canals (CAN) and otoliths are stimulated as the driver leans into the corner and the driver senses tilt. In leaning to the right he orients his longitudinal axis in the direction of the OT vector which is interpreted as the direction of upright. If the corner was prolonged the canal signal would decay and, in the absence of visual cues, the driver would lose his sense of tilt. (C) Hypothetical case in which a driver steering a straight course has an erroneous interpretation of otolith signals ($\overline{OT}$) with normal canal signals. This is similar to cornering without tilt and a possible consequence would be that he feels as though he were being pushed sideways. (D) Hypothetical case in which a driver steering a straight course has an erroneous interpretation of canal signals ($\overline{CAN}$) which could indicate tilting and turning to the right. Abnormalities of canal or otolith signals could be exacerbated during head movements giving either a heightened or diminished sensation of turn. In addition there may be combined abnormalities of canal and otolith signals.

ing is of low frequency content and out of the normal physiological range of the labyrinth. Hence vision, both peripheral and central, is of overriding importance, not only for steering but also for assessing changes in speed, and this dominance of vision obviates problems which arise from a disordered vestibular system. The vestibular system becomes more important in circumstances when vision is reduced as when driving in an empty landscape. Hence, spatial disorientation in an empty visual field is likely to be a consequence of abnormal vestibular signals, or erroneous central interpretation of vestibular information, a (fig).

The vestibular apparatus is sensitive to both angular acceleration (semi-circular canals) and linear acceleration (otoliths). If the otoliths are stimulated in isolation by the centripetal acceleration of turning they provide an ambiguous signal which may indicate either centrifugal force or tilt.³ Normally the predominate interpretation is of centrifugal force.⁴ Tilt becomes the predominate sensation if the canals are also stimulated when the head actually tilts. Hence an abnormal otolith signal could be interpreted as a force acting sideways or a lateral acceleration whereas an abnormal canal signal could give rise to the sensation of tilt or torque. Combinations of canal and otolith abnormalities are also possible. The canal and otolithic abnormalities could become manifest during natural stimulation such as turning if their abnormal function had a particular phasic sensitivity. In patients 5 and 6 the vestibular imbalance caused the drivers to veer in a similar way to a patient with an acute lateralised vestibular lesion whose gait and posture show a directional bias.

Motorway driving conditions are particularly conducive to the development of these symptoms; visual background detail is at a minimum and with the eyes looking straight ahead the "optic flow field" which moves in a series of parallel straight lines towards the driver is a weak stimulus for visual self-stabilisation;⁵ the car is generally travelling at a constant velocity with no changes in angular acceleration to stimulate the canals; the bends usually have only slight change in camber. Conversely in town, buildings, trees and traffic provide a rich visual background with plentiful references to the true vertical, and there are continual angular and linear changes in the motion of the traffic.

Hilly roads also cause particular problems for these patients. At the crest of a hill, the driver is briefly deprived of visual background. As he passes over the crest, there is a rapid decrease of gravitational force as descent commences. Thus there are large changes in the forces normally detected by the otoliths and, in these circumstances, otolith dysfunction or asymmetries may become manifest as inap-

propriate tilt sensations.

A disorder of interpretation of movement of the visual environment may add to the problems experienced by these patients. During turning, the linear optic flow field becomes asymmetrical producing a more unidirectional horizontal optokinetic stimulus. Asymmetries of the optokinetic responses and circularvection, as observed in three patients, may provoke the illusion of abnormal rotation. This would compound problems due to deranged vestibular responses as is likely in central lesions.

The symptoms described in patients 1 to 4 have some similarity to "space phobia", which has been attributed in some cases to vestibular disturbances. In case 9 of the series described by Marks,⁶ the patient felt pulled to one side and the road camber felt excessively steep. This patient had found herself on the wrong side of the road. Patient 3 of Marks and Bebbington⁷ also complained of steep road camber making driving difficult; this patient was exceptional for space phobia in not having a fear of falling. Neither of these patients had demonstrable neurological disease. Patient 4, in the present series, complained that there seemed to be an excessive gap of one or two feet between himself and the car door and this is a characteristic symptom of space phobia. It would appear probable that our patients share pathological mechanisms similar to those in the space phobia spectrum.

Cases 1-4 have all remained symptomatic but with some improvement after the first two years. The question arises as to why symptoms have persisted in these patients. Failure to compensate fully for peripheral or central vestibular disturbances can be attributed to abnormalities of cerebellar function or sensory inputs;⁸ however, there is no evidence for these in our patients. Accordingly, it may be that these patients have not adapted to the specific provocative conditions because they are non-physiological. In this respect, it is noteworthy that the patients were so distressed by their symptoms that they avoided prolongation or repetition of these conditions which would preclude full adaptation.

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